OXFORD MEDICAL PUBLICATIONS

DISEASES OF THE HEART

MACKENZIE

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BY

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SECOND EDITION

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PREFACE TO THE SECOND EDITION

THE speedy exhaustion of the first edition of this book, and the demand for its translation into a number of languages, is an evidence of the interest taken in the more recent methods of studying diseases of the heart. It is extremely gratifying to find many investigators entering a field full of promise of new and original observations, embracing every aspect of the circulatory system.

I wish to point out that, in setting forth my own experiences, I desire to keep the record of facts apart from their interpretation. For this reason the numerous figures in the text represent the actual facts as recorded by the movements of the heart and blood-vessels, and are therefore far more trustworthy than a verbal description. The interpretation of these tracings represents the present state of my knowledge. These interpretations may ultimately be proved to be incorrect, but the recorded movements will continue to serve for other and more fitting explanations.

A vast amount of clinical and experimental work still remains to be done to explain the variable changes in the heart's action due to disease, and my explanations can, consequently, only be tentative. Should other investigators prove these interpretations wrong by the discovery of new facts, I shall rejoice with them, for they will have shed fresh light, and will have reached a plane higher than I have been able to attain.

A valuable addition to the methods for clinical and experimental observation has been made by the recording of the electrical changes caused by the contraction of the heart chambers. I am indebted to my friend, Dr. Lewis, for kindly writing a short appendix on this subject.

J. M.

January, 1910.

PREFACE TO THE FIRST EDITION

In the following pages are given the results of observations on affections of the heart, made during an active practice of over a quarter of a century. As the nature of the heart affection can only be inferred from the presence of one or more symptoms, my special object has been to ascertain the mechanism by which the symptoms are produced, to find out their relationship to organic changes in the heart, to ascertain their prognostic significance, and, finally, to employ them as a guide for treatment.

This line of observation has revealed many new and unexpected features, and has necessitated the employment of special methods and the watching of individual cases for many years.

To make the results of use in practice it has been found necessary to enter into considerable detail, and it has been difficult to make the account at the same time succinct and intelligible. A certain amount of detail is necessary, yet too much would be wearisome and perhaps confusing, hence controversy has been avoided and the observations have been given with the explanation that seems most reasonable.

Many methods of examination that occupy prominent places in textbooks have been briefly dealt with or even ignored, not because I do not recognize their usefulness in certain cases, but because my object has been to deal with matters of practical value in the every-day examination of patients. It may appear that an unnecessary amount of attention has been devoted to details, many of which can only be recognized by special apparatus. But to bring conviction proof had to be elaborated. Many of the seemingly trivial signs, such as the minute differences in the size of a pulse beat, or a slight delay between the auricular and ventricular systoles, are really of vital importance in revealing

changes which have been proved to be due to very definite affections of the heart. In the same manner the study of irregularities is of the greatest service, as their presence is easily detected and their significance has never been properly understood. A close study of irregularities throws an unexpected light upon the functional derangements of the heart, and affords grounds for an intelligent diagnosis upon which a rational treatment and prognosis can be based.

The main purpose of all my work has been to obtain a guide for treatment, and my readers may be disappointed at the seeming barrenness of my work in this respect. A careful search has been made for the essential principles that should govern a rational treatment, and if only few drugs and suggestions are given, it is because the treatment of cardiac disease at present in vogue requires careful revision in the light of the more accurate diagnosis now made possible by means of the graphic method of examination.

In routine practice it is not usually necessary to take graphic records. If one is trained to make careful and minute observations by the ordinary methods, and to have these checked by graphic records, one can ultimately acquire the power of recognizing the majority of movements of the circulation without graphic records. In regard to the tracings given in this book, a selection has been made from an enormous number of observations, and are rarely exceptional, but are types of the commoner forms. The interpretation of these records may prove to be faulty, and an endeavour has therefore been made to keep the actual observation separate from the interpretation, so that if the latter be erroneous, the recorded movements may at least remain available for future workers in this field.

It was originally intended to give a more complete account of the morbid anatomy of the heart, and for that purpose Professor Keith has investigated a great number of hearts, of which I have kept careful clinical records, but the investigation, so far, has brought to both of us the conviction that before the pathology of the heart can be put on a satisfactory basis a more thorough and

minute examination of the post-mortem appearances in correlation with the clinical symptoms is necessary. The post-mortem aspect of heart disease is therefore dealt with very briefly, and my observations in this respect are intended to be suggestive rather than conclusive.

I had intended to give the means of judging the state of the heart in cases apart from those directly due to affections of the heart, as in fevers, pregnancy, surgical diseases, diseases of other organs, the administration of chloroform. This has only been carried out to a limited extent, because, when the results came to be analysed, I did not see my way clear in all cases to give satisfactory guides, so that many observations have been omitted. I hesitated to include my observations on chloroform administration, because the matter is still very vague. I feel convinced that the reason chloroform is attended with so much danger will never be solved until prolonged and painstaking investigation is made during its administration along the lines of observation detailed in this book and I leave my remarks in this unsatisfactory state in the hope that others may solve the question.

What a tool is to a workman, so should a textbook be to the busy practitioner. In cases of heart affection one or two symptoms are usually most prominent, and by giving clear definitions of the terms employed, and by arranging the index and the discussion in the text, an endeavour has been made to facilitate the rapid inquiry into the meaning of any given symptom.

J. M.

17 Bentinck Street, London, W.
September, 1908.

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DEFINITION OF TERMS

ALTHOUGH I have endeavoured in the text to explain clearly what I mean by any term, yet many terms will be employed before the reader reaches the places where they are explained. For that reason I give here a brief description of the more important terms that have been lately introduced or are not found in current literature, or which I have employed to describe conditions that have not been hitherto recognized.

a.-c. interval is the time between the beginning of the auricular and carotid waves in tracings of the jugular pulse. (Also intersystolic period described in § 163.)

Arterial degeneration is the term used to cover all forms of arterial disease. As I deal with arterial disease only so far as it embarrasses the work of the heart, I use this to avoid the more specific terms concerning which there is still so much difference of opinion.

Auricular venous pulse is the form of jugular pulsation where the wave due to the auricle is found preceding the ventricular contraction in contradistinction to the ventricular venous pulse (q.v.). Sometimes called also the normal or negative venous pulse.

Auriculo-ventricular node (a.-v. node, Knoten of Tawara) is the enlargement of the remains of the primitive cardiac tissue found in the wall of the right auricle, from which the a.-v. bundle arises (Fig. 2).

Auriculo-ventricular bundle (a.-v. bundle, Gaskell's bridge, Kent's or His's bundle). The remains of the primitive cardiac tissue which passes from the a.-v. node to the right and left ventricles.

Cardio-sclerosis. Fibrous changes affecting the endocardium and myocardium are found in the majority of cases in two groups of people, those with a history of an acute febrile affection—most commonly rheumatic fever—and those with evidences of arterial degeneration. The symptoms resulting from both conditions have a great similarity, but there are circumstances, such as age and response to treatment, that sharply separate them ; the term cardio-sclerosis, unless qualified, will always refer to the group with arterial degeneration. Fatty changes are frequently present in cardio-sclerosis, but they cannot be distinguished by clinical methods.

Conductivity is the term used by Gaskell to describe that function of the fibres of the heart muscle which conveys the stimulus from fibre to fibre. It is usually studied by observing the time between the systole of the auricles and ventricles.

Contractility is Gaskell's term for the power of contracting possessed by the muscle.

Extra-systole is the premature contraction of the auricle (auricular extra-systole) or of the ventricle (ventricular extra-systole), or both chambers together (nodal extra-systole), while the fundamental or sinus rhythm is maintained. Usually the extra-systole is followed by a long pause (compensatory pause). Rarely the premature contraction occurs between two normal beats (interpolated extra-systole).

Heart-block is the term used by Gaskell to signify the stoppage or blocking of the stimulus for contraction in its passage from the auricles to the ventricles.

Hyperalgesia. An abnormal sensitiveness to pain, shown by a painful response to such stimulation as would not normally produce pain, e. g. lightly pressing the skin (cutaneous hyperalgesia) or muscles (muscular hyperalgesia) between the thumb and forefinger.

Myogenic theory. The view that the heart muscle-fibres possess in themselves the power of originating and conveying the stimulus for the contraction of the heart, as opposed to the neurogenic theory, where it is held that the heart acts only in response to nerve stimulation.

Nodal Bradycardia is the term used for that condition of infrequent action of the heart where there is no evidence of the auricular systole between the ventricular beats and where the venous and liver pulses are of the ventricular form (see also Nodal Rhythm).

Nodal rhythm. In the majority of cases of continuous irregularity and paroxysmal tachycardia it is found that the ventricular contraction precedes or is synchronous with the auricular contraction, and hence it is inferred that the heart's contraction in these cases must originate from a point that could affect both auricle and ventricle at or about the same time. It is suggested that this point may be in the a.-v. node or its neighbourhood, and as it is important to recognize the abnormal rhythm it is provisionally distinguished by the term 'nodal rhythm'.*

Palpitation is used in a twofold sense, to describe (*a*) attacks of increased frequency of the heart's action, (*b*) the sensation by which a patient is conscious of the excited and usually increased frequent action of the heart.

Paroxysmal tachycardia is applied to a sudden increase in the heart's rate, usually followed by an equally sudden reversion to the normal. It is due to the starting of the heart's contraction at some part other than the normal. The most common form is really a transient nodal rhythm. There is also a rarer form, where the stimulus arises in the auricle. I therefore restrict the term to the increased frequency of the heart's action due to the temporary inception of an abnormal rhythm.

Primitive cardiac tissue is the term applied to the tissue in the mammalian heart which represents the cardiac tube of the more primitive vertebrates. It is shown in Fig. 2, and consists of the s.-a. and a.-v. nodes and a.-v. bundle. This view of the origin of these structures is inferred from the consideration of certain embryological, physiological, and clinical characteristics, but has not yet been fully established by embryologists.

Pulsus alternans means that form of abnormal rhythm where the radial pulse is perfectly regular but where there is an alternation in the size of the beats, and is an evidence of the failure of the function of contractility.

Pulsus bigeminus is applied to that form of pulse irregularity where every second beat is an extra-systole, and is usually smaller than the preceding normal beat. The smaller beat is invariably followed by a pause longer than the pause preceding it.

Sino-auricular node (s.-a. node). The term given by Keith and Flack to a small bundle of tissue representing the remains of the primitive cardiac tube (portion of the sinus venosus) near the mouth of the superior vena cava (1, Fig. 2).

Tonicity is the term applied to that function of the heart muscle which keeps the heart during diastole in a state of slight contraction. Depression of this function results in dilatation of the heart and of the auriculo-ventricular orifices.

* Recent electro-cardiographic observations by Lewis leads him to conclude that the disappearance of the evidence of the auricular contraction in cases of nodal rhythm is often due to the auricle passing into fibrillation.

Ventricular rhythm. This term is applied to the ventricular contractions in cases of complete heart-block. As this occurs when a lesion severs the a.-v. bundle, it is assumed that the remaining fibres of the a.-v. bundle in the ventricles start the ventricular contractions,—the rate being very slow, rarely above thirty-two beats per minute. (In some of my earlier writings I employed this term to describe the condition given under nodal rhythm.)

Ventricular venous pulse is that form of jugular pulsation in which the auricular wave disappears or coincides with the period of ventricular systole, there being no sign of the auricular wave at the normal period of the cardiac cycle. Sometimes called the positive or pathological venous pulse.

Viscero-motor reflex. The term used to describe the contraction of muscles of the external body-wall in response to a stimulation from a diseased viscus.

Viscero-sensory reflex. The sensory symptoms (pain and hyperalgesia) evoked by the stimulation from a diseased viscus of a sensory nerve in its passage from its peripheral distribution in the external body-wall to the brain.

DISEASES OF THE HEART

CHAPTER I

General Outline of the Principles underlying the production of Heart Failure

§ 1. The object of the circulation, and how it is attained.
2. The importance of the heart muscle.
3. The meaning of heart failure.
4. Reserve force.
5. Conditions exhausting the reserve force.
6. The nature of the symptoms in heart failure.
7. Methods adopted in describing affections of the heart.

As the study of the condition of the heart is approached in this book from a standpoint somewhat different from that usually taken in textbooks on diseases of the heart, I propose to give here a brief description of the principles underlying this method of study, in order that the reader may the more readily appreciate the nature and symptoms of heart failure.

§ 1. **The object of the circulation, and how it is attained.**—The object of the circulation is the supply of a constant stream of material capable of nourishing the tissues, and of replacing the loss of energy sustained by them, and the removal of such waste products as are capable of entering the circulatory channels. In order to facilitate the exchange of products between the blood and the tissues, a certain degree of slowing of the flow takes place as the blood passes through the capillaries. As a continuous pressure is required to force the blood onwards, the intermittent pressure conveyed to the blood-stream by the heart is converted by the resilient nature of the arterial walls into a constant pressure at the periphery of the arterial system. The maintenance of the arterial pressure is the outcome of the force exerted by the left ventricle, and of the resistance of the smaller arteries and capillaries. The full force of the ventricular contraction is not spent on the blood-current merely during the period of its systole. In throwing the blood into the arterial system, it does so with such force that it distends to a slight extent the larger arteries. The elastic coats of the arteries, as soon as the ventricular systole is over, compress the column of blood within them, and in this manner maintain a degree of arterial pressure during the period that the ventricle is not acting. The ventricular force

is thus stored up by the distension of the elastic coats of the arteries, and liberated during the ventricular diastole.

The principles underlying the mode of action of the left ventricle and the systemic circulation apply equally to the right ventricle and the pulmonary circulation.

§ 2. **The importance of the heart muscle.**—The heart muscle supplies the force which maintains the circulation. In the normal condition, the mechanism of the circulation is so adjusted that all parts combine to facilitate the work of the heart and to attain the object of the circulation. Any disturbance of that adjustment must at once entail more work upon the heart muscle, inasmuch as a departure from the normal means the embarrassment of the heart in maintaining the normal arterial pressure. So long as the heart can overcome the impediment, and maintain the circulation in a normal manner, no symptoms are evoked, but if the heart is no longer able to carry on the circulation efficiently, then certain phenomena at once arise, and these phenomena we call 'symptoms of heart failure'.

§ 3. **The meaning of heart failure.**—From this consideration it will be realized that heart failure is simply inability of the heart muscle to maintain the circulation, and that this failure of the heart muscle is due to a disturbance of the normal adjustment of the various factors concerned in the circulation. This disturbance may arise in a great many ways, but the end result is the same—embarrassment of the heart muscle and its final exhaustion. The heart muscle, therefore, is of such prime importance in what we call heart failure, that a close and intimate study of its properties is essential. This will be dealt with later in some detail; here I want to call attention to a feature of the heart muscle which is the essential factor in the consideration of every form of heart failure, namely, the reserve force.

§ 4. **Reserve force.**—If the part played by the heart muscle in the maintenance of the circulation and the nature of the symptoms in heart failure be considered, it will be found that the explanation of heart failure can be summed up in the general statement that heart failure is due to the exhaustion of the reserve force of the heart muscle as a whole, or of one or more of its functions. This statement may seem so self-evident as scarcely to need amplification, but, as a matter of fact, this, the essential principle on which diagnosis, prognosis, and treatment should be based, is often practically ignored. I shall make no apology for the continuous reiteration of this apparent truism, for the simple reason that in the study of all cases of heart failure the condition of the reserve force will be found to be the ultimate question.

Although we recognize what reserve force is, it is not very easy to define it in words. Physiologists do not seem to have given it that study its importance demands. Although difficult to define, its existence is proved in every movement of the body, and in every effort which is made, as it is by the possession of this quality that we are able with ease to undertake all forms of effort. The estimation of the amount of reserve present is the best test of the heart's condition. *It is the premature exhaustion of this reserve which constitutes heart failure,* and it is the heart's power to regain this reserve force on which recovery from heart failure depends.

§ 5. **Conditions exhausting the reserve force.**—I have already remarked that, in the normal condition, the adjustment of all parts concerned in carrying on the circulation is essential to efficiency. Any disturbance of the adjustment at once calls for an increased effort. Such calls are made first on the reserve force and, if persisted in, lead sooner or later to its exhaustion. These disturbances of the adjustment are extraordinarily varied in character, and may arise from any one of the factors on which the normal heart's action depends. It will be from this standpoint that the diseases of the heart will be studied, inasmuch as it is only by looking at the matter in this light that a proper perspective is obtained in regard to the significance of any abnormality. Thus, irregular action of the heart will be described from the point of view of its effect upon the efficient performance of the heart, as well as the condition producing it. Valvular defects will be studied not as a specific affection to be considered in themselves, but rather as a source of embarrassment to the heart muscle in its work. In the same manner, arterial degeneration and high blood-pressure will be considered as conditions that upset the normal adjustment of the factors that carry on the circulation. Inherent defects of the muscular wall itself will also be viewed in their bearing on the heart's efficiency. The relative efficiency of independent functions of the muscle-fibres will be kept constantly in view, inasmuch as organic lesions act deleteriously through disturbing the normal harmony of these functions. Depression of the individual functions may arise without any gross organic lesions, and lead to serious embarrassment of the circulation. While I am far from comprehending the full effect of functional depression, for the study of pure functional pathology is in its infancy, yet I hope the facts I detail may help forward a line of observation that promises much for future investigation.

§ 6. **The nature of the symptoms in heart failure.**—Briefly put, the symptoms are produced by the exhaustion of the reserve force. The first sign is invariably a subjective one, although it may for a time pass unnoticed. The patient's attention is first directed to his condition by some

disagreeable sensation evoked when he attempts to perform some act which he had been wont to do with ease, and without distress. Such a condition can be summed up as due to the too speedy exhaustion of the reserve force, and manifested by a limitation of the field of cardiac response. The sensations by which the limitation makes itself known are varied, but when carefully analysed, the true nature of the heart failure is revealed. Hence, I insist upon particular attention being paid to the patient's sensations.

The standard by which one measures the strength of the heart, or, more properly speaking, the amount of the reserve force, is in the main personal, as each individual unconsciously acquires the knowledge of what he can do with comfort, and his attention is called to the fact that his range of effort has become circumscribed. The observing physician can also detect signs in the frequent pulse and quickened respiration, out of proportion to the exertion. Other sources of information are obtained from the physical examination of the patient, not only in the heart itself, but in the condition of the circulation in other organs and tissues, as well as in the reflex protective phenomena which appear in affections of the heart as in affections of other viscera.

§ 7. **Methods adopted in describing affections of the heart.**—The ideal method in writing a book on diseases of the heart would be to take up each separate lesion, and describe the symptoms that arise in consequence of the lesion. But although this method is the one that has been attempted by most writers of textbooks, it is impracticable in our present state of knowledge. Not only are the lesions imperfectly recognized, but the symptoms themselves are usually not the outcome of the organic lesion, but result from the embarrassment of the heart induced by the lesion. Thus the symptoms of heart failure in a case of valvular disease are not produced by the valve lesion, but by the failure of the heart muscle to overcome the difficulty created by the damaged valve, and this failure may be brought about in a variety of ways by the greater exhaustion of one function than of another,—sometimes, for example, it is the contractile power of the heart that gives way, sometimes the tonicity ; so that we get a variety of symptoms corresponding to the exhaustion of different functions. But these functions may become exhausted from other causes than valvular lesions, and similar symptoms may therefore be induced by organic lesions of great diversity. Another reason for not following this ideal method is that it is extremely rare that the heart is the seat of only one lesion, or of a lesion limited to one particular part. Acute affections of the heart, for instance, are often described as endocarditis or pericarditis, because there happens to be a marked systolic murmur or a pericardial friction sound. But in

addition to these audible signs, there may be presented a great number of symptoms such as dilatation of the heart, irregular action of the heart, and so forth, and these are given as being symptoms of the endocarditis or pericarditis. As a matter of fact, these symptoms are not the outcome of endocarditis or pericarditis, but of a myocardial affection, and the attempt to give a precise description of the symptoms pertaining to each organic lesion has led writers to ascribe phenomena to lesions with which they have no connexion, with the result that the symptomatology of heart affections is to-day confused and contradictory.

In searching for a reason for this confusion, I am inclined to attribute it to the fact that the human mind attaches the greatest importance to that class of phenomena which most strongly affects the senses. Many of the really vital and all-important symptoms are so subtle and so slight that it takes the most careful methods to detect them, whereas a roaring murmur or an irregular pulse thrusts itself upon our attention. The result is that the subtle signs are ignored and all the stress is laid upon the murmur and irregularity. Murmurs and irregularities, therefore, have come to occupy a far more important place in the cardiac symptomatology than their true significance merits.

These considerations have led me to adopt a method which I hope will place the symptoms in a proper perspective. Recognizing the fact that the attention of the physician or the patient is drawn to the heart by the appearance of one or more symptoms—and it is only by the recognition of the symptoms that the diseases can be inferred,—I write this book from the standpoint of the symptoms. So far as I can, I describe each symptom, give its physiological meaning, and the various pathological conditions with which it may be associated. Then I try to estimate its significance. From the data thus obtained, conclusions are drawn as to prognosis and treatment.

CHAPTER II

Fundamental Functions of the Heart Muscle-cells

§ **8. Myogenic doctrine.**—While it would be somewhat beyond my province to enter into a discussion of the question whether the heart contracts in response to a nerve stimulus, or in response to a stimulus developed in the muscle-cells, it is very necessary, in order to comprehend the meaning of the signs and symptoms that arise in affections of the heart, to appreciate the phenomena which are associated with the contraction of the muscle-fibres. The conception of the meaning of the heart beat, which we owe to Gaskell, has been supported by careful and minute analysis of the functions of the normal heart muscle-fibres, and the interpretation of the symptoms of heart affections in the light of this knowledge has revealed so clearly their true meaning as to revolutionize the study in the human subject. Even if the 'Myogenic doctrine' be ultimately proved untenable, the investigations carried out in its support have added so much of value to our knowledge of the heart's action that its conception will ever be associated with a great stride forward, not only in physiology, but also in the recognition and treatment of diseases of the heart. It is just possible that the two opposing doctrines—Neurogeny and Myogeny—may be reconciled along the lines I suggest in the following brief summary of the main points. I do not enter into much detail, but give such salient points as are necessary to appreciate the explanation of the symptoms I give in the course of this work. For fuller details the reader is referred to Gaskell's article on 'The Contraction of the Cardiac Muscle' in Schaefer's *Text Book of Physiology*.

From the consideration of the physiology of the cell, it may be said that every function possessed by a cell in the fully developed state exists partially developed in the primitive form. However specialized the function

may be, nervous, muscular, or secretory, these functions can be referred back to some property possessed by the primitive cell. The cells which constitute the primitive structure of the body all start equally endowed, and it is by a gradual process of specialization that each takes on its peculiar function, while as it acquires a high degree of specialization it gradually loses those functions it no longer exercises. The functions possessed by the primitive cells can therefore be deduced not only from the results of direct observation, but from the specialized functions of the more differentiated tissues, even if these functions be so highly developed that they bear little resemblance to those found in the primitive cell. For instance, the excitability of the cell may become so specialized as to be responsive only to certain stimuli, as heat, pain, light, sound, while all its other functions are apparently lost. If one looks at the functions of nerve and muscle cells in the fully developed state, it seems at first sight difficult to realize that they had originally identical functions. The primitive cells, from which the heart developed, had all the same characteristics, yet in their final evolution they present widely divergent characteristics both in appearance and in function. That some such modification does occur must be inferred when we witness the change in function that takes place in the evolution of the heart from the primitive cardiac tube. I therefore suggest as a working hypothesis, that in the evolution of the heart muscle-fibres certain functions of the primitive cell were retained, some of these being more developed than others according to the duties the fibres had to perform, so that while they have come to resemble muscle-fibres, they nevertheless retain in a varying degree some functions which are highly specialized in the nerve-cell.

The special functions which Gaskell has demonstrated are five in number, namely :—

(1) The power of producing a stimulus which can excite the heart to contract—stimulus production.

(2) The power of being able to receive a stimulus—excitability.

(3) The power of conveying a stimulus from fibre to fibre—conductivity.

(4) The power of contracting when stimulated—contractility.

(5) The power to retain a certain amount of contraction even when the active movement has ceased—tonicity.

§ 9. **Stimulus production.**—From this point of view it is assumed that the heart muscle-fibres, if supplied with appropriate nutriment, possess a power of internally secreting a material which is capable of stimulating the fibre to contract. This material is being continually secreted, and during a pause in the heart's contraction, accumulates in the heart cell. When sufficient has been stored to excite the heart to contract, the whole store

is used up in stimulating the muscle-cell. Immediately after the contraction the store again begins to accumulate until sufficient has been produced to excite the heart to further contraction. This function, being continuous in its action, cannot control the rhythm of the heart, but by its co-operation with the other functions a rhythmical character is given to the accumulation and destruction of this material.

§ 10. **Excitability.**—The heart muscle depends for its contraction upon its power of receiving a stimulus—that is, upon its excitability. Immediately after the heart has been stimulated to contract, the fibres are no longer capable of further stimulation, excitability has disappeared, and the fibres are in what is called the refractory stage. The excitability begins at once to be restored, and increases very rapidly during diastole. This is demonstrated by the heart being susceptible to weaker stimuli the longer the time since the previous contraction. So long as the heart is capable of contracting, the rate of the heart depends upon the functions of stimulus production and excitability, and when the conditions are normal the equal action of both functions—the stimulus material being renewed at a uniform rate, and the restoration of the excitability taking place uniformly—a regular rhythm of the heart's action results. Under normal circumstances, therefore, the heart's rate and rhythm are dependent upon the integrity of these two functions.

§ 11. **Conductivity.**—In a mass of primitive cells the individual cell has a power of passing the stimulus on to neighbouring cells. This function of conductivity is possessed by the heart muscle-fibres, for the stimulus is passed on from cell to cell from the point where it originated. The possession of this function by the muscle-fibres of the heart gives them a character which is typical of certain forms of nerve-fibres, but in the heart this is not so highly developed as in the specialized nerve-fibre, the conduction of the stimulus not being so rapid in all cases, and much more easily exhausted. Like every other function of the heart, it is entirely abolished after it has been exercised, and it returns gradually. The rate an impulse travels also varies in different fibres of the heart. Some fibres, such as the more recently developed contractile fibres of the auricle and ventricle, conduct the stimulus with much greater rapidity than the fibres which convey the stimulus from auricle to ventricle.

§ 12. **Contractility.**—The power of contraction is the most evident of all the functions of the heart. By the co-ordinated contraction of the fibres of the different portions of the heart, the circulation is maintained. After a contraction, this function is completely exhausted, and the power returns very gradually. Within certain limits the strength of the contraction

depends upon the length of the period of rest preceding the contraction, the function gathering strength during quiescence.

§ 13. **Tonicity.**—The functions of the heart muscle do not differ, except in degree, from those of other muscular structures, and as tone is a very characteristic property of muscular tissue, it is certain that the heart muscle will possess it, and it is shown by the fibres not relaxing to their full length during diastole. On account of the rapid action of the heart it is not easy to demonstrate this function. Gaskell[107] has shown that the degree of relaxation depends on the amount of tone present, and that certain drugs increase or diminish the amount of relaxation. Thus antiarin, veratrin, and digitalis prevent the relaxation of the heart muscle in the frog so that the heart remains longer in the condition of complete contraction, the relaxation gradually becoming less and less, till finally it is almost impossible to recognize individual beats. On the other hand, solutions of lactic acid and muscarin produce the opposite effect, the heart becoming more and more relaxed, the contraction diminishing in size till the heart stands still in diastolic relaxation. Just as certain portions of the musculature have certain functions more highly developed, it would not be unreasonable, as Gaskell says, to expect that different parts of the heart should vary in their tonicity. That this expectation is justified will be manifest when all the symptoms of heart failure are considered, and it is only on recognizing that the heart possesses this very important function that we can understand some of the most significant features of heart failure.

§ 14. **Co-ordination of functions.**—When the complicated action of the heart is considered, it will be readily recognized that though all the fibres may be endowed with these functions, a further specialization is a necessity for the co-ordinated movement of the different parts of the heart. If all the fibres were equally endowed, then all would contract simultaneously. As it is, certain fibres at the venous end have the functions of stimulus production and excitability more highly developed than others, so that after a period of rest the contraction starts in them. The stimulus then proceeds to adjoining fibres in such a manner that the process of stimulation and contraction sweeps through the whole heart, with the result that the different chambers and the different parts of each chamber contract in that order and degree necessary to the efficient carrying on of the circulation. If any other part of the heart be rendered more excitable than the venous end, then the contraction starts there. As the stimulus then does not sweep through the heart in the normal manner, the heart's action is less efficient, and heart failure may thus arise.

§ 15. **Characteristics of the functions of the heart muscle-fibres.**—

While the heart may be said to carry on its work in consequence of its possession of these functions, there are other important features in each which have a practical bearing on the symptoms of diseases and the principles of treatment. The integrity of these functions depends on the supply of suitable nutriment, and sufficient time of inactivity to recover after their exercise. When a contraction takes place, all the functions have been exercised to the full extent of the power possessed by the fibres at the moment of stimulation. No heart-cell exhausts only a portion of its function; when stimulated, it uses all the energy which it possesses (all or nothing). For a brief period after their exercise, the functions cease to exist: recovery, however, begins at once during the period of rest, and each function in time regains its strength, so that, within certain limits, the longer the delay, the more complete is the recovery, and the more efficient is the subsequent action. It is by fully appreciating the effect of rest and proper nourishment that we gain the best conception of the principle that should underlie our treatment of heart failure. While all the functions when exercised use all the force they possess, they nevertheless manifest a quality whereby they can respond, under certain circumstances, with a greater activity. Thus the rate may be suddenly increased, and at the same time the stimulus passed from the auricle to the ventricle with increased rapidity, and the contraction be executed quicker. These changes are to a great extent under the control of the nervous system, but they imply a quality possessed by all these functions which is of vital importance to us in the study of heart failure. For, as I have already remarked, it is this power of responding to effort that gives us the clue to the real state of the heart.

When one reflects that all the fibres of the heart are not equally endowed with the same functions, and that all the functions may not always be exposed to an equal strain, it is but reasonable to conclude that conditions may arise where they are unequally affected. As a matter of fact, this is what commonly happens, and it is an interesting and important question to consider in each case of heart failure what functions are specially at fault. The significance of this question was demonstrated when Wenckebach[223] showed how the irregular activity of the various functions or of the various parts of the heart were made manifest by certain characteristic arrhythmias. Following up the idea of exhaustion or over-excitability of individual functions, I have sought to connect many of the symptoms of heart failure with these functions. While I do not say that my conclusions are invariably correct, they have led to some definite results of the very highest importance, and it is along these lines that advance in our knowledge will likely follow for some time.

CHAPTER III

Development, Anatomy, and Physiology of the Heart

While I take it for granted that the reader is familiar with the ordinary textbook description of the anatomy and physiology of the heart, there are some recent investigations which have an important bearing on the clinical investigation of heart disease which need to be considered. For that reason I give here a brief résumé of certain points necessary for the appreciation of clinical signs.

§ 16. **The primitive cardiac tube.**—In an early stage of the embryo's development, the heart appears as a tube. The veins from the body unite into a common cavity—the sinus venosus—at the posterior end of this tube. In the course of development this tube becomes bent upon itself, and from it, later, pouches develop which ultimately become the auricles and ventricles—the original tube still persisting and connecting them (Fig. 1). As development proceeds, the sinus venosus loses its distinctive feature as a separate structure to become incorporated in the termination of the superior and inferior vena cavae, and a strip of the right auricle between the orifices of those two vessels, and the coronary sinus. Probably the terminal part of the pulmonary veins is also derived from the sinus. At the same time the original cardiac tube ceases to exist as a tube but it is inferred that it persists as the connecting medium between auricles and ventricles in the shape of a band of peculiar fibres—the a.-v. bundle (Fig. 2). It thus loses its function as a propelling organ, which is taken up by the auricles and ventricles.

§ 17. **The functions of the primitive cardiac tube.**—The functions of the primitive cardiac tube and its representative in the mammalian heart need to be carefully studied, as the appreciation of the nature of its functions has the most important bearing on many cases of heart failure. Its peculiar properties have been studied most fully in the heart of the frog, also

in the toad, tortoise, crocodile, &c., but, so far, only to a slight extent in the mammalian heart. In the lower vertebrates the primitive tube is still recognizable in the sinus venosus, auricular canal, and aortic bulb (Fig. 1). It has been found that the posterior end of this tube is the most excitable portion, and in consequence of this the heart's contraction starts at the sinus venosus. The remainder of the tube also possesses the faculty of starting the heart's contraction, only in a less degree than the sinus. If, however, any part of the tube be rendered more exeitable than the sinus the

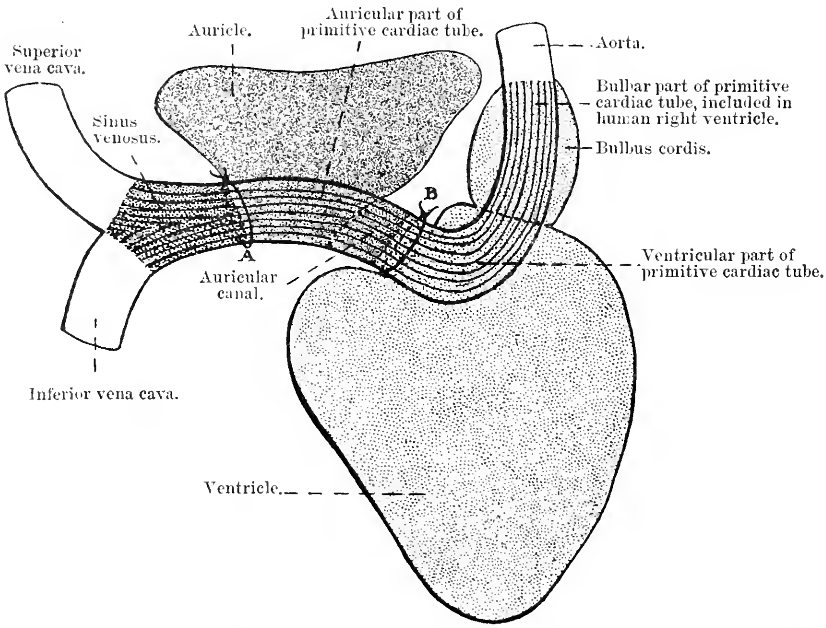

FIG. 1. Diagram of the primitive vertebrate heart, showing the development of the auricle and ventricle from the primitive cardiac tube. (Keith.)

contraction starts from that part. The peculiarity of the primitive cardiac tube and its relatively greater excitability than the auricular or ventricular tissue is brought out in the following experiment by Gaskell[123]: 'Touch the auriculo-ventricular ring of muscle (i.e. the primitive cardiac tube) with the slightest stimulus, immediately a series of rhythmical contractions occurs. It is most striking to see, after removal of the septum, how every portion of auricular and ventricular tissue can be explored up to the very edge of the ring, without obtaining more than a single contraction, while immediately the needle touches the muscular ring a series of rapid contractions results.'

The possession of the power of independent contraction by the separate

portions of the heart is brought out in the Stannius experiments. When a ligature is applied or a cut made between the sinus and auricle of a frog's heart (*A*, Fig. 1), so as to sever completely the connexion, the sinus continues beating, and after a pause of varying duration the auricles and ventricles begin to beat at a rate different from, and independent of, the sinus. This rate is slower than the sinus rate, and sometimes the auricle contracts before the ventricle, sometimes the ventricle precedes the auricle, and very rarely they contract simultaneously. (Engelmann [122]).

If a second Stannius ligature be applied between auricle and ventricle (*B*, Fig. 1), the sinus and auricle continue beating, and after a period the ventricle takes on its own rhythm, slower than, and independent of, the other portion of the heart.

§ 18. The remains of the primitive cardiac tube in the mammalian heart.—The sinus venosus, which in the primitive heart normally originated the stimulus for contraction, has no representative as an independent structure in the human heart. Morphologists recognize that it has become incorporated in the great veins near the heart, and physiologists observed that the peculiar functions of the sinus venosus were found over a somewhat wide distribution. Normally, the contraction starts with such uniformity in a number of places that no one place can be pointed out with certainty as the starting-point, though the mouth of the superior vena cava seems to lead the contraction. In certain experiments dissociation can be shown, and independent pulsations start at the coronary sinus and pulmonary veins. From this it is inferred that in these separate places some remains of the original sinus venosus persist, the possession of which endows them with the power of starting independent contractions. Until very recent times no definite remains of the sinus venosus had been found. Keith and Flack [123] have described lately a small node of tissue—the sino-auricular node (1, Fig. 2)—at the mouth of the superior vena cava. This tissue consists of fine delicate pale fibres faintly striated, in which branches of the vagus and sympathetic nerves terminate, and is supplied with a definite artery. These observers consider that this node of tissue represents a portion of the sinus venosus, from which, probably, the heart's contraction starts. Similar tissue has so far not been found in the other veins. It is possible that some fibres may be scattered about, but, not being grouped into a definite node, they are not capable of being differentiated from the muscular fibres by which they are surrounded. The further remains of the primitive cardiac tissue are probably found lower down, arising in the right auricle and passing across the a.-v. septum to be distributed in the ventricles. Although the presence of this bridge was inferred in the mammalian heart by Gaskel, it

was not demonstrated until 1893, when first Stanley Kent[305], then His (junr.)[296], described its presence and both experimentally demonstrated some of its functions. A full description of its structure and ramification was given in 1906 by Tawara[323], whose elaborate examination of the tissue has been of the greatest interest and importance. This bundle rises from a node of tissue—the a.-v. node (2, Fig. 2)—situated in the right auricular wall near the mouth of the coronary sinus, and Cohn[236] has recently worked out some of the details connecting it with the muscle-fibres of the auricle. The bundle passes over the auriculo-ventricular septum below the central fibrous body and under the septal cusp of the tricuspid valve. While on the septum it divides into two, one branch passing into the left ventricle and the other into the right. In the right ventricle it continues its course as a narrow rounded bundle in the muscle-wall of the heart, till it approaches the apex, where it divides into numerous fine threads, terminating in the muscle-fibres. In the left ventricle the bundle rapidly widens out into a thin band which passes down to the apex splitting into fine branches.

The character of the fibres constituting this bundle varies. The fibres of the a.-v. node are of the same delicate nature as those of the sino-auricular node. As this bundle passes into the ventricle the fibres change, becoming thicker, the greater part of the cell body being undifferentiated protoplasm only faintly striated at the circumference and containing a large nucleus. In their final distribution they are recognized as being the fibres described long ago by Purkinje. Another peculiarity of this bundle is that it is isolated from the structures in which it is embedded by a fine sheath of connective tissue. There are numerous nerve structures in the a.-v. bundle which have been specially studied by Gordon Wilson[329]. He finds numerous ganglion cells, abundant nerve-fibres, an intricate plexus around the muscle-fibres of the bundle, and distinct vaso-motor nerves. Finally, it is mainly supplied by a special branch of the right coronary artery, a fact of some significance in the pathology of the heart.

§ 19. **Functions of the primitive cardiac tissue in the mammalian heart.**—The functions of this bundle have been only partially explored experimentally, though a fair inference of its functional action can be made from clinical symptoms. Kent[305], His (junr.)[296], Erlanger[239], Hering[292], and others have demonstrated that it conveys the stimulus from auricle to ventricle, for compression of the bundle interferes with the conduction, while section stops all connexion, and the ventricle contracts at a rate quite independent of the auricular rate, as after the second Stannius ligature. An exactly similar experience is met with as a result of disease in man.

But, as has been seen, the primitive cardiac tube also possesses the power

of originating the stimulus for contraction, and there is every reason to suppose that if it be rendered more excitable the remains of the primitive cardiac tissue in man will start a contraction independent of the sinus rhythm. In the neighbourhood of the node there are certain centres which when stimulated modify the heart's action. Thus MacWilliam [129] has shown that a small area in the auricle here, when stimulated, causes marked slowing of the ventricular rate. Whether this is a portion of the vagus mechanism or some

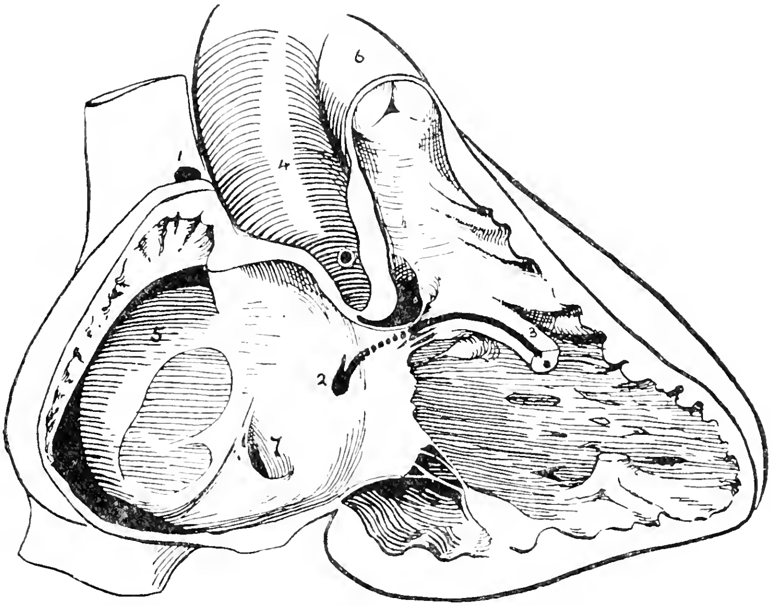

Fig. 2. Section of a heart, exposing the septal wall of the right auricle and ventricle and showing the position of a portion of the remains of the primitive cardiac tube. 1. Superior vena cava above the sino-auricular node. 2. Auriculo-ventricular node (Knoten of Tawara), from which the auriculo-ventricular bundle arises. The interrupted part represents the main bundle, and the continuation to 3 is the right division, where it is shown in the cut moderator band. 4. Aorta. 5. Right auricle below the superior caval orifice and taenia terminalis. 6. Pulmonary artery. 7. Opening of coronary sinus. (Keith.)

portion of the primitive cardiac tissue has not been determined. Lohmann [310], by irritating the a.-v. node, or its immediate neighbourhood, caused the starting of the auricular and ventricular contractions together. In the human subject I have attempted by a process of reasoning to show that extra-systoles may arise in this tissue, and that the continual irregularity recognized so frequently in the later stages of rheumatic heart affection and cardio-sclerosis, may be due to the inception of the rhythm of the heart by the irritated primitive cardiac tissue probably in or about the a.-v. node.

§ 20. **Functional anatomy of the heart.**—I mention here a few of the more important features connected with the heart as a muscular organ which are of importance in this clinical study. These are based on Keith's [156] descriptions, who has demonstrated that the heart is built of muscle bundles whose points of origin and insertion are as definite as those of skeletal muscles, and whose functions can also, to a great extent, be inferred with equal certainty. Naturally the separation of the different muscle bundles is not so complete as in skeletal muscles, there being a continuous connexion between neighbouring fibres, so that they pass gradually from one system into another.

In order to appreciate how the muscle-fibres act, and also to understand the changes that result from an increase in the size of the heart, it is necessary to comprehend how the heart is fixed in the thorax.

The pericardium is a fairly unyielding structure fixed firmly above to the cervical fascia and below to the central tendon of the diaphragm. The aorta and the great veins, where they penetrate the pericardial sac, receive a covering from it, and these may be regarded as fixed points. The lungs also may be considered as ligaments which attach the base of the heart to the whole of the chest-wall.

The contraction of the heart starts at the mouth of the great veins. It has hitherto been assumed that regurgitation from the auricle was prevented by the contraction of the circular fibres at the mouth of the veins. Keith shows that these are too weak for the purpose, except around the coronary sinus, and that regurgitation back into the superior vena cava is prevented by the contraction of the broad band of muscle which sweeps over the roof of the auricle—the taenia terminalis. In its contraction this muscular band shuts off the vein from the auricular cavity. As the taenia is attached at the orifice of the inferior vena cava and is carried over the roof of the right auricle, it also aids in the closure of the inferior vena cava and the pulmonary veins. The pressure of the blood in the inferior vena cava apparently renders a perfectly competent closure of the inferior caval orifice unnecessary.

Arising from the taenia are a number of other muscular bands, the pectinate fibres, which pass across the auricle to be fixed in the auriculo-ventricular septum. In their contraction, in addition to assisting the taenia in emptying the auricle, they pull up the ventricles by reason of their insertion into the a.-v. septum (*A*, Fig. 3).

The ventricles have themselves no real fixed point, but depend for security on the fixation of the vessels at the base of the heart. Immediately below the origin of the aorta in the heart is the central fibrous body, which is really a tendon for much of the ventricular muscle. The remainder of

the ventricular muscle is inserted into the a.-v. septum. The other fixed point during contraction is the apex. It becomes a fixed point in virtue of the peculiar arrangement of the muscles here constituting the 'whorl'. A great number of fibres from different parts of the heart converge here and

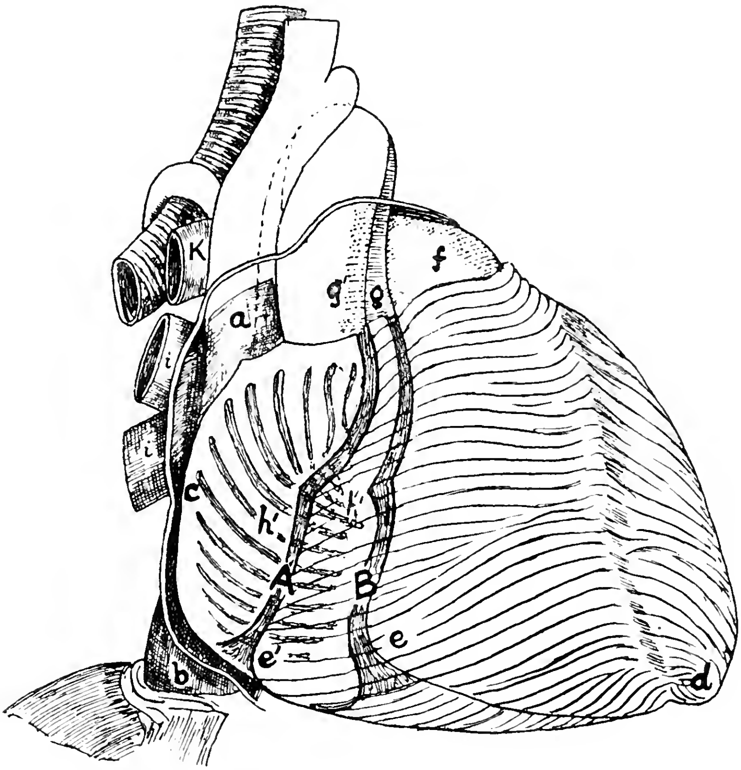

FIG. 3. Sketch of the heart to show the movements of the auriculo-ventricular groove during auricular and ventricular systole. *A*, position of groove when pulled upon by the contraction of the pectinate fibres of the auricle; *B*, position of the groove when pulled upon by the contraction of the ventricular fibres. During the diastole the groove occupies a position midway between *A* and *B*; *a*, sinus portion of superior vena cava; *b*, inferior vena cava; *c*, is on the taenia terminalis; *d*, apex of the heart; *f*, pulmonary artery; *i*, *i*, pulmonary veins; *k*, pulmonary artery. During ventricular systole *g′* is pulled to *g* and *e′* to *e*; *h′*, musculi pectinati during auricular systole; *h″*, during ventricular systole. (Keith.)

mutually support one another in contraction, the result being a fixed point from which the fibres can exercise traction in different directions. With the onset of the ventricular contraction the apex of the heart rises up and presses firmly against the chest-wall. As no shortening takes place between the apex and the aorta, all the parts of the heart are drawn towards the line between apex and aorta. Hence it is that while during ventricular

contraction the apex is pushed forwards, all the other portions of the cardiac surface are dragged inwards, and this explains the variation in the appearance of a cardiogram taken from different parts of the front of the chest. At the same time, as Chauveau[139] and Keith[156] have shown, the ventricular fibres inserted into the a.-v. septum not only diminish the size of the ventricle, but enlarge the size of the auricle by dragging down the a.-v. septum —a fact of some importance in the production of the venous pulse (*B*, Fig. 3).

§ 21. **The nerve supply of the heart.**—The independent functions of the muscle-fibres enable the heart to execute its movements independently of any nervous intervention, nevertheless nervous influences have a powerful effect in modifying the activity of the various functions of the fibres. The nerves of the heart are usually described as being of three sorts, namely, (1) inhibitory fibres passing to the heart, (2) accelerator or augmentor fibres also passing to the heart, and (3) depressor fibres passing from the heart.

The inhibitory fibres are derived from the spinal bulb by the internal branch of the spinal accessory nerve, and pass down in the vagus and reach the heart by its cardiac branches. The effects of the vagus upon the heart are varied. On division of one vagus little effect may result. If both vagi be cut the frequency of the heart is much increased. If the vagus be stimulated the result is curiously varied. Its action may be said in a general way to depress the functions of the heart muscle-fibres, but it does not do so uniformly in all cases. It usually acts first on the excitability of the heart or on the stimulus production, so that the whole heart becomes slower in its action, or the whole heart may stand still for a brief period. With stronger stimulation, it may act on the conductivity of the fibres joining auricles and ventricles, and depress this function so that the ventricle fails to respond to every auricular systole. Underlying this seemingly uncertain action of the vagus is a principle which is of great importance in diagnosis and in treatment, namely, that if there be depression of one function of the heart, vagus stimulation is liable to seize upon that function and increase the depression. The fact that depressed functions are more susceptible to vagus stimulation seems to be the reason for some of the discordant results arrived at by experiments.

The accelerator fibres belong to the sympathetic system and have their origin in the spinal cord, passing out of the cord by the white rami communicantes to the upper four or five dorsal nerves. They pass upwards to the inferior cervical ganglion ; from thence they pass to join the cardiac fibres of the vagus, and so reach the heart. Stimulation of these fibres increases the rate of the heart, sometimes very considerably, and

according to Roy and Adami [178] they increase the strength of contraction and output as well, and hence have been assumed to contain augmentor fibres.

The depressor fibre is a definite nerve which arises in the heart, and, passing upwards, joins the vagus nerve and so reaches the bulb. Stimulation of the peripheral part of the cut nerve has no effect, but stimulation of the central end causes a fall of blood-pressure through reflex action on the medulla oblongata.

Afferent fibres.—Such is a brief résumé of the heart nerves described in physiological textbooks, but the whole matter is not included here. There is a great field of evidence which is entirely lost to the physiologist, but which is open to the clinician. The personal sensation of the animal cannot be communicated to the experimenter, nor can the changes in sensation that result from stimulation of the cardiac nerves be ascertained. In dealing with the symptoms of heart affections it will be shown that there is unquestionable evidence of a system of nerves passing from the heart to the spinal cord and the bulb. The principles involved in the production of these symptoms have not been thoroughly appreciated, and I shall enter more fully into their explanation when dealing with the reflex or protective phenomena (Chapter VI).

Cardiac ganglion cells.—The nerve-fibres of the a.-v. bundle have been referred to on p. 14. The ganglion cells in the heart have been studied by Gaskell [123]. He says, 'What, then, are the ganglion cells in the heart? What function do you attribute to them? That is a question which I am ready to answer, and to answer with confidence, as follows: The ganglion cells in the heart are part of the great group of ganglion cells which are situated on the course of the small-fibred efferent nerves supplying the viscera. These cells form the outlying vagrant groups of nerve-cells which are known by the name of the sympathetic and cerebro-spinal ganglia. In the case of the heart, the ganglion cells are the cells belonging to the small-fibred efferent cardiac fibres of the vagus, just as some of the cells in the ganglion stellatum and in the inferior cervical ganglion are the cells belonging to the small-fibred efferent cardiac fibres of the augmentor nerve. There is no more reason to assign special functions to these cells than to any of the other peripheral efferent nerve-cells. They are cells connected only with the inhibitory fibres of the vagus, and as such are simply part and parcel of the mechanism of inhibition, just as the corresponding cells in the ganglion stellatum are simply part and parcel of the augmentor mechanism.'

CHAPTER IV

Preliminary Examination of the Patient

§ 22. **The patient's appearance.**—Before entering upon the physical examination of the patient, the physician ought to obtain a clear and comprehensive appreciation of the patient's own sensations. I therefore wish to insist upon the importance of the preliminary examination, for it often happens that a thorough appreciation of the patient's own experiences is of more value in arriving at a correct estimation of the heart's efficiency than the most elaborate methods of physical examination. The attempt to appreciate the patient's condition should begin when first he presents himself before us. On his appearance in the consulting-room, his bearing, his gait, the condition of his respiration, the colour of his face, any nervous peculiarity in his manner of speech and behaviour, and so forth, should be noted. If he is in bed, note the position he assumes, and any change in his colour or respiration in response to such exertions as talking or turning over. By habit, one unconsciously notices these things, and as the examination proceeds first one trivial matter, then another, may arise, which helps materially in guiding the examination and in forming the final opinion.

§ 23. **The patient's sensations.**—After ascertaining his age and occupation, ask the patient to describe the chief symptoms from which he suffers. In his replies, insist upon these being precise and definite. When he refers to his feelings, get him to indicate the location by placing his hand upon the region; otherwise, proud of his small anatomical knowledge, he will attribute his sensations to his viscera; and here I may add a warning to the physician not to make a note of the sensations by attributing them to any viscus: thus a pain should not be put down as felt in the heart, stomach, liver, or lungs, but only in the region of the body indicated by the patient, for it will probably be found on later examination that the pains are not felt

in the viscus. In other words, he should make no notes that might prejudge the nature of any symptom until he has before him all the evidence.

Having obtained from the patient an account of the symptoms from which he suffers, a brief inquiry should be made regarding the other viscera, as to the state of the digestion, the urinary secretion, the breathing, &c. Particular inquiries should always be made in regard to sleep, for in many patients the want of sleep induces a breakdown, and the power of a patient to pass a night in comfort or otherwise, often gives important indications. The condition of the brain, its power of attention, memory, dreams, and so forth, are often of material value.

§ 24. **The patient's history.**—After this, I usually inquire into the past history for other illnesses, the nature of employment, whether there is worry, excessive indulgence in tobacco or alcohol. Guided by the information thus obtained, I return again to the main complaints, and inquire when they began to appear, and what induced their appearance. In all cases it is necessary to find out what degree of exertion the patient can undertake with comfort. As I have said, heart failure resolves itself into the question of the amount of reserve force, and consequently it is at this juncture that we begin to find out that amount; therefore the factors that produce the first sign of discomfort need the most detailed inquiry. The sequence of the phenomena as they arise is most instructive. The patient has often only a confused notion of the order of appearance of the sensations, and the situation in which they are felt, and it may be necessary to postpone a final opinion until a second visit, and the patient should be instructed to note, should occasion arise, the individual sensations, their location, and order of appearance. On a subsequent visit the account may be totally different, but often one made with great precision. It is sometimes surprising to observe that a patient, after an attack of agonizing pain, has but a very dim idea in what region it was felt. Inquiries should be made as to the presence of less obtrusive symptoms, which experience tells us to expect, but which the patient often ignores, unless his attention is drawn to them. Thus when he complains of a pain in the chest, is it associated with a sense of constriction, or after it passes, is there a desire to micturate ? Many patients have bad dreams and even delusions, and, as Head points out, the latter are never elicited unless carefully inquired after.

§ 25. **The chief complaints.**—The chief complaints from which patients with heart failure suffer are as follows :—

Breathlessness.—This first appears on the patient making some effort which was not previously attended with discomfort, or dyspnoea more or

less extreme appears without any apparent cause. The various forms of respiratory trouble are described in Chapter V.

The sense of exhaustion.—Many people complain of feeling simply 'done up'. The sensation may come on after a bodily or mental effort, and particularly after some excitement. It may be a feeling of exhaustion in general, or it may be located in some definite region, as the epigastrium, or across the chest. It is a symptom common to trivial and transient conditions of heart exhaustion, and to conditions of the utmost gravity, in the latter case usually associated with other phenomena. A curious sense of exhaustion referred to the legs in walking is occasionally complained of by elderly people. They feel, after walking a short distance, as if they were walking in water, or forcing the legs through some viscous fluid. A similar condition is found in patients suffering from heart-block, and some of these are never really comfortable unless in the recumbent position.

Pain may appear in all degrees of intensity, and be of slight or serious import, the most severe not necessarily being the most serious, nor the slightest being of the least significance. It may come on with exertion, and at once bring the patient to a standstill. It may not appear until some hours after the exertion which induced it. It may be felt in various regions of the chest, or arms, or epigastrium, or neck. Its starting-point and radiation should always be noted, as it follows frequently very definite lines, which reveal its true origin. A pain may also be felt in other regions, as over the liver, when there is enlargement of that organ due to heart failure.

A sense of constriction or oppression of the chest is a fairly common accompaniment of pain, but it may appear independently of pain, and be so severe as to compel a cessation from all effort. The symptoms associated with pain are described in detail in Chapters VI and VII.

Palpitation is the consciousness of the impact of the heart against the chest-wall. The heart is frequently also quickened in rate, though not necessarily so. In neurotic people, the hammering of the heart against the chest-wall is most distressing.

Consciousness of irregular action of the heart.—Allied to this is the consciousness of irregularity. The most common form of irregularity is that where there is an occasional long pause or intermission, the next beat of the heart being strong, giving rise to an unpleasant sensation. So severe may the shock from this strong beat be that in some people it induces a feeling of exhaustion. Instead of being conscious of the strong beat after the pause, the patient may be aware of the pause, and feel as if the heart had stopped, or he may feel some curious sensation as a 'catch in his breath'. Irregularity

may give rise to a slight fluttering sensation in the chest. When the heart becomes continually irregular for a short or a long period, this fluttering makes the patient conscious of the abnormal action (see Chapter XX).

Haemorrhages.—Rupture of an artery due to high blood-pressure acting on a degenerated artery may give rise to cerebral apoplexy, bleeding under the conjunctiva, from the nose, &c. Nose bleeding is not infrequent with disease of the aortic valves, and in women attacks of bleeding from the nose are liable to occur at the beginning of the menstrual period. Haemoptysis may occur when the lungs are engorged or when there is a rupture of a blood-vessel.

Cerebral Symptoms

There are a number of symptoms produced by the brain dependent on the inefficiency of the circulation. I exclude from consideration here the symptoms due to lesions of the blood-vessels, as rupture. The symptoms induced more directly by affections of the heart are caused by a diminished supply of blood reaching the brain, and the nature of the symptoms depends on the extent of that diminution and the period during which the diminished supply lasts.

Dizziness or giddiness.—The first stage of transient cerebral anaemia is shown by a sensation of giddiness. It is seen most characteristically in elderly people (particularly in tall people), when there is arterial degeneration. The attack usually comes on when the individual makes a sudden change in his position, as in rising from a couch. The attack may be limited to a passing sensation of giddiness, or the individual may reel and stagger and clutch at some object for support, or he may fall. In walking, a transient dizziness may come on and the individual may stagger for one or two steps. The liability to these attacks varies at different times—periods of liability varying with periods when the tendency disappears. The attacks occurring in elderly people have no special prognostic significance, as I have known persons exhibit them over a long period of years and live to over eighty years of age. Exactly similar attacks may occur where the heart fails to send sufficient blood into the brain, as in paroxysmal tachycardia, where the transient increased rate is accompanied by a diminished output from the heart.

Loss of consciousness.—Syncopal attacks or fainting, when the patient becomes limp and unconscious, occur with a diminished supply of blood to the brain. This can be brought about in a variety of ways, and is generally due to an alteration in the heart's action. I have made observations and taken tracings of several patients during syncopal attacks, and

have found a variety of conditions present. The most common has been a slowing of the heart-rate, with great weakness of the pulse, so that only a slight tracing was obtained by the sphygmograph. In one case the heart was beating with great rapidity, but the pulse beats were small and the patient lay unconscious for nearly half an hour. In another case the heart became very slow in its action and irregular with beats of varying strength. The more frequent forms of syncope are preceded by a preliminary sensation of extreme weakness and loss of sight. 'All became dark' is a very frequent expression made by the patient after recovery.

There is one form of loss of consciousness which is met with in elderly people. The attacks come without warning and are of momentary duration, resembling *petit mal.* The individual may be sitting at his desk, when his head suddenly drops on the desk, or he may be standing or walking when he suddenly falls. Consciousness at once returns and he is surprised to find himself in a strange posture. There is generally present extensive arterio-sclerosis in these cases, with an irregular heart due to extra-systoles. I am disposed to consider the attacks due to several of these extra-systoles occurring in succession, so that there is a temporary cessation or a diminution in the amount of the blood supplying the brain. Similar attacks occur in the early stages of heart-block, but tracings of the jugular pulse enables one to distinguish the condition.

Adam-Stokes syndrome.—When the blood-supply of the brain is completely arrested, loss of consciousness results, and, if prolonged, epileptiform contractions of the muscles of the body may result. The condition of heart affection where this is seen most typically is in heart-block—when the auricle continues to beat at a normal or quickened rate and the ventricle stands still or beats at a very slow rate. But identical cerebral phenomena may arise from other conditions when the ventricular contraction is too slow, as in the condition I describe under the term 'nodal bradycardia' (p. 337), or in the long standstill of the whole heart, probably due to vagus influence, as described by Laslett [308].

The symptoms due to a diminution of the blood-supply varies according to the degree of the cerebral anaemia. Patients suffering from one of the conditions just mentioned may show a variety of symptoms, as a feeling of dizziness, a brief loss of consciousness, or a prolonged loss of consciousness with twitchings of the muscles or even convulsions. These varying degrees depend on the frequency of the ventricular systole, the milder phenomena being shown when the ventricular standstill is prolonged, or when the contractions are at rare intervals. Dr. O'Connor, in the case of nodal bradycardia, described on p. 342, noted that his patient might have twenty

or thirty brief attacks of loss of consciousness in one day, and on several occasions when talking to his patient he observed him suddenly become pale and lose himself for a few seconds, the attack exactly resembling one of *petit mal*. He has had his finger on the patient's pulse on several of these occasions and felt it disappear. During longer pauses in the pulse this patient had numerous epileptic convulsions.

Barr [257] has noted in a case of heart-block that convulsions occurred when the pulse disappeared for twenty seconds. In tracings taken from the same patient, pauses of twenty-one seconds and over were followed by convulsions. With prolonged disappearance of the pulse and unconsciousness, convulsions do not always occur, and in the same individual during long pauses there may be no convulsions, or there may be slight muscular twitchings or wide-spread convulsive movements, under apparently similar circumstances.

How long it is possible for the pulse to disappear and recovery of consciousness to be complete has not been determined in the human subject, nor can the observation of the pulse alone be relied upon to give information. Leonard Hill has pointed out that the merest trickle of blood may keep the brain intact, and the fact that in heart-block the auricles are beating may account for a certain amount of blood reaching the brain. That the auricle can send blood into the arterial system can be inferred from the fact that its contraction does affect the radial pulse, as shown by Figs. 122–4, pp. 183–4.

Hallucinations.—When the heart is failing the blood-supply of the brain is not fully maintained and certain symptoms may be detected. Thus the patient can maintain a mental effort as in reading, or in writing, only for a short time, becoming easily fatigued. The memory for recent events becomes impaired, and what the patient may read is imperfectly retained. In more severe cases hallucinations may appear and may take various forms. The patient may imagine that some one is hiding behind a door and though in conversation admitting the unlikelihood of such a circumstance, yet when left alone the hallucination returns with a sensation of terror. Or the patient may wake up and imagine he sees objects, as an arm projecting from the ceiling, or the sound of some one on the stair, or of soldiers marching past in the street. Head [15] has dealt very fully with this matter, and points out that the patient usually conceals these hallucinations until he is directly questioned concerning them.

CHAPTER V

Respiratory Symptoms

The respiratory symptoms arising in the course of heart affections are so numerous, and the conditions causing them so multifarious, that it is impossible to deal exhaustively with this subject. The factors concerned in the production of any one of the forms of respiratory trouble are often difficult to recognize, and I shall not attempt any strict analysis of these factors, but limit myself to the more apparent clinical forms that accompany heart affections.

§ **26. Breathlessness, or air hunger.**—There is normally a ‘ desire to breathe ’, and under certain circumstances this may be intensified so that there is a ‘ hunger ’ for air. The sense of air hunger compels laboured breathing so that the term ‘ breathlessness ’ includes the subjective sensation and the objective symptom. The centre for respiration in the medulla is influenced by its blood-supply, and its nervous connexion with the periphery. A free blood-supply is necessary to supply oxygen and remove the carbonic acid, and an insufficient interchange of these gases is betrayed by an increased activity of the centre, thus giving rise to breathlessness. The respiratory centre is also receptive to peripheral stimulation, as from sensory nerves of the skin and from cardiac and pulmonary nerves.

An absence of the sense to respire may occur for a short period as in the apnoeic stage of Cheyne-Stokes respiration. John Hunter [338] gives a curious account of an attack from which he suffered, when he lay pulseless but conscious ; observing he did not breathe, and had no desire to do so, he thought he must die if he did not breathe, and by an effort of will

continued to breathe until his pulse returned and the automatic respiration was established.

§ 27. **The sense of suffocation.**—This is difficult to describe, and is referred usually to the upper part of the chest and throat. The mechanism is obscure. It is a frequent accompaniment of heart affections, from temporary weakness of the heart to the gravest forms of heart disease, and its significance may be slight, or in serious heart trouble it may be a very grave sign.

§ 28. **Inability to stop breathing.**—I have been struck by the fact that while taking tracings of the jugular and other pulses many patients are unable to stop breathing. When asked to hold their breath, they will shut their mouths, but are unable to stop breathing through the nose. If the nose be pinched, they then complain of distress (air hunger). This condition only arises in extreme heart failure, and as the heart regains strength we may recognize the fact by the ability to hold the breath.

§ 29. **Quiet, rapid breathing, free from distress.**—In many cases of heart failure, when the patient is lying quiet, no definite symptom can be found save that of a respiration more rapid than normal. There is no distress, and the symptom is apt to be overlooked. Sometimes I have failed to recognize it until I have come to analyse tracings where the respiratory movements had been taken. Where, as sometimes happens, a patient describes certain feelings (as angina pectoris) which we are bound to recognize as cardiac in origin, but where physical examination reveals no sign of mischief, the recognition of more rapid respiration may prevent us from viewing too lightly the condition of the patient. This symptom is of the greatest importance in patients suffering from affections of the heart, particularly in mitral-stenosis, in exhausting diseases like typhoid fever, and in all conditions that compel the weakly, and particularly the elderly, to lie on their backs, as e. g. when suffering from a broken leg. In such cases the lying in bed, while favouring the work of the left ventricle, embarrasses the work of the right by restricting the respiratory movement. As a result of this restraint of the ribs, the breathing becomes shallow. The effect is to retard the flow of blood through the less mobile part of the lungs, and in consequence stasis at the base of the lungs results (see § 190).

§ 30. **Continuous laboured breathing.**—This occurs characteristically, at the beginning of what is called ‘ sudden failure of compensation ’. The patient cannot lie down, but sits up breathing in short quick gasps. The slightest exertion, such as is entailed by changing to a more comfortable position, immediately aggravates the distress. This form of breathing is best seen in cases of dilatation of the heart with a rapid irregular pulse,

and, in fact, the whole respiratory distress is often the outcome of the heart taking on the nodal rhythm which is manifested by the irregularity (Chapter XX). With slowing of the heart's action and other evidences of cardiac improvement, the respiratory distress gradually disappears, but is liable at first to be brought on readily by a slight exertion.

§ 31. **Laboured breathing brought on by exertion.**—This is a symptom common to a great many affections besides heart troubles, but its association with a weak heart is so common that it should at all times lead to an inquiry as to the condition of the heart. It may occur with every form of heart affection. It is of very great use in estimating the strength of the heart, as its appearance indicates in a rough way the degree of exhaustion. The earlier it appears, the less the reserve power of the heart. On the other hand, improvement may be indicated by the gradual return of the ability of the patient to undertake with comfort greater exertion. The forms of exertion that induce it are very puzzling. Some patients can lift great weights, and pursue the hard work associated with their calling, yet cannot walk up a slight hill without complaining of being short-winded ; or they suffer in the same way on going into the cold air. Some can cycle in comfort, while others cannot cycle, but can walk with comfort on the level. I cannot explain these, and other peculiarities, but note them as clinical facts.

§ 32. **Attacks of breathlessness (cardiac asthma).**—A form of respiratory distress which comes on usually in the night, sometimes suddenly arousing the patient from sleep, has received the name of ' Cardiac Asthma '. An attack of this sort is sometimes the first serious sign of heart trouble, though on inquiry an account can usually be obtained of a period prior to the attack when there was a distinct limitation of the field of cardiac response. The patient may have gone to bed in his usual health and according to his usual custom, and after three or four hours' sleep he is awakened with a feeling of suffocation, and an intense desire to breathe deeply. He sits up in bed, and breathes in deep and laboured fashion. A sense of great prostration may add to his suffering. Wheezing sounds may appear in the chest, and he may cough up some frothy phlegm. The attack may last for half an hour or longer ; then the breathing becomes quieter, and he is able to lie down, though he keeps starting up, and finally assumes a position with his head and shoulders raised, passing the remainder of the night in uneasy slumber. Once these attacks begin they are apt to continue, and the nights of the patient often become periods of great distress. The fear of the attack may keep the patient awake, and should he drop off to sleep he awakes with a start at the first sign of embarrassed breathing.

The class of case which shows this condition most characteristically

is the elderly, and those who suffer from cardio-sclerosis. Generally they have a high blood-pressure, and the heart is usually regular except for the presence of occasional or frequent extra-systoles. In these cases we sometimes find the best examples of the pulsus alternans. The cause of this form of asthma is not quite clear. I have found the great distress induced by this condition in many cases insusceptible to treatment, but lately I have been giving massive doses of oxygen, in some cases with very gratifying success (see p. 278).

§ 33. **Cheyne-Stokes respiration.**—In all cases of heart affection where there is reason to suspect cardio-sclerosis, or where there is a high blood-pressure from such causes as arterio-sclerosis and Bright's disease, and where we get a history of breathlessness, quiet observation should be made when the patient is perfectly still, and nothing attracting his attention. Under such circumstances, the patient's breathing may occasionally be found to take on a peculiar rhythmical character. Instruction should also be given for the attendants to note the character of respiration during sleep.

The character of the breathing is a rhythmical variation in the size of the respiratory movements—a gradual passage from a state of complete or almost complete cessation to a condition of breathing where the respiratory movements are deep and laboured (see Figs. 4 and 5, Plate I). When the breathing gradually slows and ultimately stops, the patient's mental condition frequently becomes a blank, and is accompanied usually by twitching of some muscles, especially to be noticed in those of the hand and arm. With the resumption of the breathing, he wakes, and may resume a conversation that he had been pursuing before the slowing of the breathing set in.

Usually no material change can be detected in the action of the heart during the diverse phases of respiration. Sometimes, however, certain changes may appear in the pulse coincident with the respiratory phases. One patient with a sclerotic heart (Case 23, Appendix V) had shown a well-marked pulsus alternans for many months (Fig. 249), and towards the end of his life he developed Cheyne-Stokes respiration (Fig. 6, Plate I). During the respiratory phases there was a distinct increase in this form of irregularity in the radial pulse. I took his blood-pressure during a period of Cheyne-Stokes respiration, and found that it fell 5 to 10 mm. Hg. during the apnoeic stage. This variation can be made out in the pulse tracing in Fig. 6, Plate I, where it shows a greater amplitude of beat, and lowering of the base line during the fall of blood-pressure, i. e. during the apnoeic period. Gibson has also described a fall of blood-pressure during the apnoeic stage. In Fig. 7, Plate I, there is a tracing from a patient who had hiccough as well

as Cheyne-Stokes respiration, and Cushny[389], commenting on this case before the Physiological Society, remarked, 'During the intervals of apnoea, the rhythm of the hiccough was more rapid than during the periods of active respiration, but the violence of the spasms seemed somewhat less.

'The physiology of hiccough is still obscure, and we are not aware of any record of the movements having been taken previously. It is generally stated to be a reflex movement of the diaphragm, originating from some abnormal irritation of the stomach. If the respiratory centre is involved in the reflex, as is generally held, it must have remained excitable by nervous impulses in this case when the normal chemical stimulus failed to induce respiration. The slowing of the hiccough rhythm during the periods of active respiration is striking, and may perhaps be analogous to the interference of two reflexes with a final common path (Sherrington); here only one afferent stimulus is of nervous origin, however, the other being chemical.'

Usually the patient is unaware of the occurrence of this periodic respiration, and it causes him no distress. On the other hand, it may occasion acute distress: the patient may drop off to sleep, and this form of respiration gradually appears, and during the apnoeic stage the patient awakes with a most distressful sensation of suffocation. So terrible is this that patients extremely exhausted may spring suddenly out of bed in an extremity of terror.

The great majority of cases of Cheyne-Stokes respiration are brought on by, or associated with, cardio-sclerosis, arterial disease, and high blood-pressure. It disappears with the sudden fall of blood-pressure due to dilatation of the heart. It may be made temporarily to disappear by massive doses of oxygen; or on the inhalation of small quantities of CO_2 (Pembery[420]). Haldane and Poulton[490] have produced the condition by forced breathing, and conclude that the periodic breathing is due to the 'disappearance of the (indirect) excitatory effects of want of oxygen in the respiratory centre'.

In the great majority of cases of arterio-sclerosis and Bright's disease it is usually the beginning of the end, the patients dying within a few months or weeks, or even a few days of its onset. In a few cases I have seen it appear two or three years before death. It calls for no special treatment save when the apnoeic stage produces the terrible sense of suffocation; then morphia or chloral generally relieves this feature without altering the respiration.

Cheyne-Stokes respiration must be kept distinct from certain other forms of periodic respiration. Hibernating animals show a form of

periodic respiration, also some children during sleep. It appears also in some cases of tubercular meningitis.

Loss of consciousness does not always ensue during the apnoeic stage of Cheyne-Stokes respiration, as shown by the following instance. I saw in consultation a clergyman, aged fifty, who had a paralytic seizure two years previously, resulting in an ordinary hemiplegia. When I saw him he was lying in bed quite conscious, extremely prostrate, with a pulse rate of 180 per minute, with typical periodic respiration. During the apnoeic stage he was quite conscious, and could talk intelligently, his voice was faint and reedy in quality, diminishing in loudness towards the end of his remarks. His medical attendant told me he had suffered off and on from this condition for three years. This case corresponds to a description given by John Hunter [338], who says, ' A gentleman had a singular asthmatic affection, and his breathing gradually stopped and again gradually recurred, but became violent, and thus constantly and alternately held two or three minutes, and when the breathing ceased yet he spoke although but faintly.'

§ 34. **Slow respiration.**—There is a number of individuals in whom the respirations during rest are much slower than normal, 7 to 10 per minute. The slow respiration induces an irregular action of the heart, and is described in Chapter VIII. The condition is probably due to vagus stimulation, and I have been able to produce it artificially by the administration of digitalis.

§ 35. **Pulmonary haemorrhage.**—Bleeding from the lungs is not an infrequent complication in heart failure. It is most commonly seen in the terminal stages of arterio-sclerosis, with the fall of blood-pressure that indicates dilatation of the heart and stasis of blood in the lungs. In these cases the expectoration is either blood-stained or it is almost entirely composed of lumps of dark clotted blood. *Post mortem* there are usually found several hard patches of ecchymosed blood at the base of the lungs. In the young with mitral stenosis, profuse haemoptysis may occur, and is generally a sign of extreme gravity.

In other forms of heart affection, haemorrhage more or less free may occur, and give the patient considerable relief, and be followed by no serious consequence. In fact, if we find the patient on the whole with a fair amount of reserve force, whatever the nature of the heart lesion, there need be no immediate alarm from a free haemoptysis when due to cardiac trouble.

A serious form of haemoptysis arises from a pulmonary infarct, or a pulmonary apoplexy ; in these cases the symptoms are extremely varied in severity. Thus I have seen patients with phlebitis of the veins of the leg, or after confinement, die in a few minutes with symptoms of great respiratory

distress. In some cases I have seen the most intense dyspnoea with gradual loss of consciousness go on for four or five hours, and then suddenly the dyspnoea ceases and the consciousness returns. After twelve hours the patient has expectorated large quantities of pink-stained jelly-like mucus. I have also seen cases of pulmonary infarct, as after a fracture of the tibia, where the only evidence was the expectoration, for three or four days, of dark-coloured blood, in small quantities at a time, followed by complete recovery. Presumably the difference in the symptoms in these cases depended on the size of the infarct or the extent of the apoplexy.

§ 36. **Acute suffocative oedema of the lungs.**—A peculiar form of oedema, of which I have only seen a few cases, is that in which the patient is suddenly seized with breathlessness, usually during the night, followed speedily by the welling out of the mouth and nose of large quantities of froth. Usually the patient succumbs within an hour of the commencement of the attack. The conditions giving rise to it are obscure, and it occurs in a great variety of cases. An excellent account of a case, and the literature on this subject, are given by L. Williams [440].

CHAPTER VI

Reflex, or Protective Phenomena

§ 37. Classification of symptoms in visceral disease.
38. Insensitiveness of the viscera to ordinary stimuli.
39. The mechanism by which pain and other reflex phenomena are produced in visceral disease (the viscero-sensory reflex).
40. The purpose of visceral reflexes.
41. Why pain is referred to regions remote from the organ.
42. The relationship of the heart to sensory nerves.
43. The viscero-motor reflex.
44. Vagal sensory reflex.
45. Conditions in which angina pectoris is induced.
46. Conditions giving rise to attacks of angina pectoris.
47. Association of angina pectoris with exhaustion of the muscle of the heart.
48. Association of angina pectoris with impairment of the function of contractility.
49. Summation of stimuli as a cause of angina pectoris.

§ 37. **Classification of symptoms in visceral disease.**—When the symptoms of visceral disease are carefully analysed there appears a great similarity in the nature and origin of certain of them, which permits of their division into three groups ;—first, symptoms due to changes in the organ itself—in the case of the circulatory system, changes in the movements of the heart and blood-vessels ; second, symptoms observed in remote organs and tissues which suffer indirectly from the primary lesion, for instance, jaundiced skin in liver affections, uraemic convulsions in renal disease and dropsy, oedema or albuminuria in heart failure ; third, reflex or protective phenomena, which form the subject of this chapter.

While the character of the symptoms in the first two divisions depends on the size and specific function of each organ, the reflex phenomena produced by all organs have a great resemblance. This is particularly the case with all the hollow muscular organs. They have similar origins and their reflexes are similar in character, and are only modified by the special development of each. In spite of the seeming dissimilarity in the form and function of the digestive tube, uterus, ureter, and heart, they are fundamentally the same, and the reflexes associated with them are of a like nature. Hence the nature of obscure symptoms in affections of the heart may be revealed by an inquiry into the meaning of similar phenomena presented by these organs, which have at first sight so little affinity with it.

§ 38. **Insensitiveness of the viscera to ordinary stimuli.**—In order to appreciate the reflex symptoms in visceral disease we must keep in mind the functions of the nerves that supply them.

The nerve-supply of the body is included in the two great systems, the autonomic and the cerebro-spinal. The autonomic includes the whole of the sympathetic nerves, and certain cerebral nerves, of which the vagus is the only one that concerns us here.

The tissues and organs supplied by the nerves from the autonomic system are not endowed with sensation in the sense in which the term is used in regard to tissues supplied by the cerebro-spinal nerves. The skin, muscle, and other tissues of the external body-wall are readily sensitive to all forms of stimuli that produce such sensations as touch, pain, heat, and cold, whereas the viscera supplied only by the autonomic system are totally irresponsive to such stimuli. Thus such organs as the heart, stomach, bowels, liver, kidney, can be cut, torn, burnt, and no sensation elicited. Yet, as we know, pain of a most excruciating character can arise from visceral affections. Harvey[12] describes how he touched the exposed heart of the son of Viscount Montgomery and found it without sensation. Surgeons in pre-chloroform days incidentally refer to the insensitiveness of the viscera. Thus Richerand[44] describes how, in operating in the neighbourhood of the heart, he found the pericardium insensitive, and I have repeatedly verified this observation in cases where the ribs have been resected. The following experience illustrates the insensitiveness of the viscera, and at the same time affords an insight into the manner in which visceral pain arises.

I had occasion to resect the bowel in a conscious subject under the following circumstances. He had an umbilical hernia, and had worn for years a pad tightly pressed over it, until the skin had ulcerated. The ulceration had finally penetrated into the bowel, and his food was discharged through the fistula. It was resolved to resect the bowel, but the patient would not be anaesthetized. Observing that the skin was already ulcerated, and that the tissues forming the external wall were not very sensitive, so that the abdominal cavity could be opened with little pain, I reasoned that the after-operation could be performed painlessly. It turned out as I had expected, and I was able to break down numerous old and recent peritoneal adhesions, to detach them from the liver and bowel, to resect a piece of bowel and mesentery, and to stitch these structures without the patient experiencing the slightest sensation. But I found that he occasionally groaned with pain when I was not touching him, and watching to see the cause I found that the upper part of the resected bowel, which was laid on one side in a warm aseptic cloth, occasionally passed into peristalsis,

contracting from a wide tube to a thick fleshy rod ; when this happened the patient groaned with pain. I asked him where he felt the pain, and he passed his hand invariably over the umbilical region. I started the peristalsis several times by slightly pinching the bowel, and each time the patient felt the pain. Here before my eyes was the cause of the pain, and the seat of origin of the pain was at least twelve inches away from the part in which the pain was felt.

From this experience the following deductions can be made : first, that the stimuli that produce pain and other sensations in the external body-wall are not adequate to produce these sensations when applied to the viscera ; second, that violent contraction of non-striped muscular fibres can produce pain, but the region in which the pain is felt is different from that in which the contracting muscle lies.

This isolated experience has been confirmed by many other observations I have made, and in part confirms the experiments made by Haller[10] more than 150 years ago upon animals, when he showed that they were indifferent to severe mutilation of their viscera, so long as the external body-wall was not interfered with.

These experiences compel one to look for an explanation of the production of visceral pain other than that which suffices to explain its production from the stimulation of the external body-wall, and the following explanation seems satisfactorily to account for the matter, and to explain the peculiar nature of the sensory phenomena that arise in visceral disease.

§ 39. **The mechanism by which pain and other reflex phenomena are produced in visceral disease (the viscero-sensory reflex).**—When a nerve that terminates in a sense organ is stimulated in any part of its course from the periphery to the brain, a stimulus is conveyed to the brain of a kind similar to that induced when the peripheral end-organ is stimulated. Thus the stimulation of any part of the optic nerve or auditory nerve gives rise to the sensation of light or of sound. In the same manner, if a sensory nerve be stimulated in any part of its course through the brain, spinal cord, or trunk of the nerve, the resultant sensation is referred to the peripheral distribution of the nerve in the external body-wall.

Fig. 8 is a diagram representing the brain and spinal cord (*S.C.*) with a sensory nerve (*S.N.*) passing from the skin (*Sk*) through the spinal cord to the brain. A stimulus applied to the skin, or to the sensory nerve between the skin and the cord, or to the sensory nerve in the cord, gives rise to a sensation referred by the brain to the portion of skin innervated by the nerve, though the stimulus may have affected the nerve after it had left the skin. In the diagram, a viscus (*H*) is represented, and its nerve

(*Sy. N.*) is seen passing to the spinal cord. In the normal processes of life, a stream of energy from the viscera is continually passing by the afferent

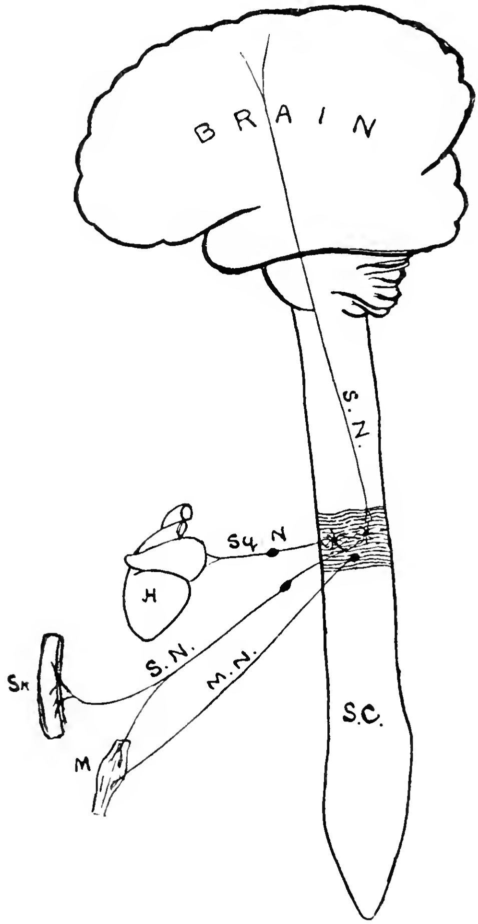

Fig. 8. Diagram showing the mechanism producing visceral pain. From the viscus *H* an abnormal stimulus is conveyed by the sympathetic nerve (*Sy. N.*) to the spinal cord *S.C.* On reaching the cord the abnormal stimulus spreads beyond the sympathetic centre and affects nerve-cells in its immediate neighbourhood. The cells so stimulated react according to their function, the sensory causing a sensation which the brain recognizes as pain, and refers to the peripheral distribution of the sensory nerve (*S.N.*) in the skin (*Sk*) or muscle (*M*), the motor (*M.N.*) producing contraction of the muscle (*M*). The abnormal stimulation may leave a portion of the cord abnormally irritable (shaded portion), so that the tissues supplied by nerves from that portion of the cord are hyperalgesic, and attacks of pain, as of angina pectoris, are more easily provoked.

nerves to the spinal cord, and continuously playing upon the efferent nerves that run to muscles, blood-vessels, and so forth. These processes are

conducted so that they give rise to no appreciable sensation. If, however, a morbid process in a viscus gives rise to an increased stimulation of the nerves passing from the viscus to the spinal cord, this increased stimulation affects neighbouring centres. If it excites the sensory nerve represented in the diagram as passing from the skin to the brain, the resulting sensation will be referred by the brain, not to the viscus, but to the peripheral distribution of the sensory nerve. Thus it was that when, in the course of the operation which I have described, the bowel contracted, the resulting pain was referred not to the bowel, but to the peripheral distribution of the sensory nerves in the region of the umbilicus—thus the pain in visceral disease is seen to be of a reflex character—*a viscero-sensory reflex.*

In the diagram there is shown a motor nerve (*M.N.*) arising in the cord and passing to a muscle (*M*). The stimulus from the viscus (*H*) passing into the spinal cord, may excite the cells of origin of the motor nerves, with the result that the muscle is stimulated to contract; hence we get the *viscero-motor reflex.* This reflex is best seen in affections of the abdominal viscera when the abdominal wall becomes hard, due to tonic contraction of the muscles.

When a portion of the spinal cord becomes violently stimulated by reason of a visceral affection, that portion of the cord may remain for a lengthened period in an over-excitable state, so that all the nerves that arise from this portion of the cord may be much more easily stimulated. This can be demonstrated in many cases of visceral disease by the hyperalgesic state of portions of the external body-wall, and by the exaggerated motor reflexes. In such instances, stimulation of the skin or muscles, as by light pinching between finger and thumb of such weak force that normally it would only result in the sensation of touch, is felt by the patient as pain. Light stroking of the skin with a pin head may be felt as pain, and produces very readily a strong reflex contraction of the muscles whose nerves arise from the same portion of the cord. A further result of this *irritable focus* in the cord is that the visceral stimulation more readily induces pain, and not only may the original attacks of pain (as in angina pectoris, renal and biliary colic) be more easily induced, but stimuli from other sources may induce the attack of pain. Thus in cases of gall-stones with hyperalgesia of the external body-wall over the region of the liver, the ingestion of the food into the stomach may give rise to much pain in the hyperalgesic tissues.

§ **40. The purpose of visceral reflexes.**—Reflexes have been studied with the greatest minuteness, and the work of Sherrington [49] demonstrates the extraordinary variety and complexity of the spinal reflexes, but their purpose has been to a great extent overlooked. The main purpose of many

reflexes has been either to remove the body from the reach of injurious influences, or to interpose a firmly contracted muscle between the agent threatening the injury and the organ. It may even be true, as Herbert Spencer suggests, that the evolution of muscles and the segmentation of the body was due to the necessity of protection when the external body-wall changed from an insensitive hard carapace to a sensitive mobile covering. The way that the protective mechanism comes into play is by exalting the sensitiveness of reflexes. Thus if an organ suffer injury, as by inflammation, the surrounding portion of the external body-wall immediately has its protective functions exalted. This is usually accomplished by increased sensitiveness of the skin and underlying structures, so that touch arouses at once a strong and vigorous contraction of the protective muscles. These muscles themselves become much more sensitive to pain, and, as painful stimuli are the most provocative causes of reflexes, acutely tender muscles may remain permanently contracted.

To illustrate the meaning and purpose of visceral pain and allied phenomena, a case of gastric ulcer may be taken. The ulcer may be situated on the posterior wall and at the cardiac orifice of the stomach, while the sensation of pain is referred to the epigastrium. Here the skin and muscles may be found exceedingly tender to touch and light pressure. The reflex contraction of the underlying recti muscles is extremely lively and powerful. If you try gently to palpate the stomach, these muscles at once become so strongly contracted that it is utterly impossible to feel what is underneath. What is nature doing ? Manifestly it is interposing a most efficient barrier between the intruding hand and the diseased organ—the whole being a protective mechanism. Had the stomach only been sensitive, the hand would have reached the stomach, but by the reflex mechanism, the external body-wall is made sensitive, and the powerful reflex contraction of the muscles effectively guards the stomach from injury. The same protective mechanism is involved in joint-disease. Thus the shoulder-joint may be found immobile. Put the patient under chloroform and all the muscles relax, and a grating may be detected on moving the joint. It is evident that here the injured joint required protection, and the muscles responded to their first and primitive duty.

Pain itself, Hilton pointed out, has the same purpose,—protection. It commands at once cessation of any action that induces it, and this protective function is seen nowhere more clearly than in heart affections.

§ 41. **Why pain is referred to regions remote from the organ.**— In a great many instances the pain is referred to situations remote from the organ giving rise to it. Thus the pain of biliary colic may be felt in the

epigastrium, the pain of renal colic in the testicle, the pain of heart affections in the arm.

The reason for this is that in the course of development the tissues, that in a low scale of life immediately covered the organ, have been displaced. Thus, the pain felt in the testicle in renal calculus is due to the fact that in its journey down to the scrotum the coverings of the testicle receive a twig from the first lumbar nerve, from which the kidney is also innervated, and when the centre of this nerve in the spinal cord is stimulated, as in renal calculus, the pain is felt in the testicle, and exquisite pain may be elicited on pressing the testicle. On the other hand, one never finds the skin of the scrotum hyperalgesic in these cases, but only the deep covering of the testicle, because the scrotum is supplied by the sacral nerves.

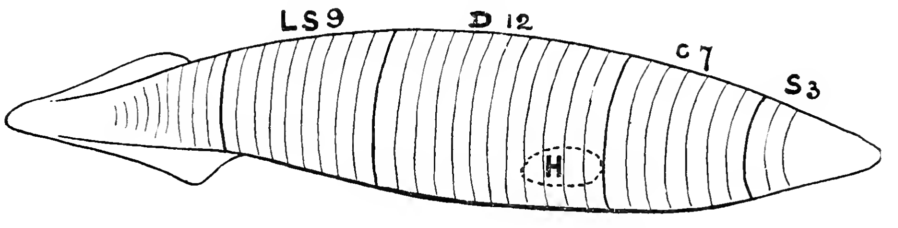

Fig. 9. Diagrammatic representation of a primitive vertebrate animal to show the distribution of the sensory nerves. For clearness of comparison the number of segments is represented to be the same as in man, and the heart occupies the same position. Each nerve is shown as limited in its distribution to one segment (after Ross).

§ 42. **The relationship of the heart to sensory nerves.**—In order to appreciate the mechanism of the pain felt in affections of the heart, the manner in which the upper dorsal nerves come to be distributed should be borne in mind. Ross [45] has pointed out that in the primitive vertebrates, before the development of the limbs, each spinal nerve is distributed segmentally round one half of the body (Fig. 9). The upper dorsal nerves are therefore entirely distributed over the body-wall and to the tissues covering the heart. The upper limbs, as they bud out from the trunk in their development, drag with them away from the trunk portions of the cervical and upper dorsal nerves, so that parts of the first and second dorsal nerves are distributed to the ulnar border of the forearm and inner surface of the upper arm. Thus, a stimulus originating in the heart and affecting the cord area of the first and second dorsal nerves, would be felt as pain in the lowly vertebrate over the heart, whereas in man it would be felt in the upper arm or in the forearm (Fig. 10).

This peculiar distribution of the nerves to the chest and arm, resulting from the development of the limbs, provides a unique field for observing

the mechanism of many nerve processes. The character of the field can be seen in the distribution of the shaded area in Figs. 10, and 12 (p. 51), which represent, the one the distribution of the eruption in a case of herpes zoster affecting the spinal ganglia of the eighth cervical, first and second dorsal nerves, and the other the area in which pain was felt, and in which the skin was hyperalgesic in a case with the symptoms of angina pectoris. The existence of these and similar associated areas has been demonstrated by a variety of methods clinically, experimentally, and by dissection.

FIG. 10. The dotted area shows the distribution of an eruption due to herpes zoster in the peripheral distribution of the eighth cervical, first and second dorsal nerves. There was also a small patch of eruption over the left scapular spine.

We know that the efferent cardiac fibres of the sympathetic pass out of the spinal cord with the upper dorsal nerves. Although it is impossible experimentally to demonstrate the region of the spinal cord to which the afferent nerves pass, clinical observation provides ample evidence, and this evidence demonstrates that the afferent nerves pass into the cord at the same regions from which the efferent pass out.

The usual description given of the pain in angina pectoris is that it is felt in the heart and shoots into the arm, or that there are two pains, a local pain in the heart, and a referred pain in the arm. If, however, a careful analysis be made of all the symptoms present, facts will be found that practically demonstrate that in angina pectoris there is but one kind of pain, and that its production is in accordance with the law I have attempted to establish, namely, that it is a *viscero-sensory reflex*. It is not in every case that one is able to demonstrate the truth of this hypothesis, but the conclusions drawn from observations made in suitable cases may legitimately be applied to others. Shortly, these observations are, that the pain in the very gravest cases may be felt in regions distant from the heart, as in the left arm; that this pain is identical in character with that felt over the heart; that the pain may originally start in parts distant from the heart and gradually approach and persist over the heart, and lastly, that the tissues of the external body-wall over the heart may be found extremely hyperalgesic

after the pain has passed away. From this last fact it is inferred that, inasmuch as the seat of pain corresponds to the region of hyperalgesia, therefore the pain was felt by the hyperalgesic nerves. To assume otherwise is to ignore a principle that explains satisfactorily the sensation of pain wherever arising.

§ 43. **The viscero-motor reflex.**—So far I have dealt mainly with the viscero-sensory reflex, but no less striking evidence can be found of the viscero-motor reflex among the group of symptoms included in the term 'angina pectoris'. Some would limit the term 'angina pectoris' to that class of cases where, in addition to the pain, there is a sense of constriction in the chest, amounting to the sensation at times as if the chest were gripped in a vice, or as if the breast bone would break. I am convinced that these sensations arise from spasm of the intercostal muscles, and correspond to the hard contraction of the flat abdominal muscles in affections of the abdominal viscera. If one watch a case of what is called 'muscular rheumatism', where the intercostal muscles are affected, and where these muscles are stimulated by the slightest movement to violent cramp-like contractions, one cannot but be struck by the resemblance to the description given by the 'gripping' sensation experienced by patients suffering from certain affections of the heart. I have watched the attacks in such cases, and could find no difference between them and those where the sense of contraction was the chief symptom in heart disease.

The viscero-motor reflex may be present alone, or, as is more commonly the case, it may be associated with the pain. The purely viscero-motor reflex is seen best in the elderly, where it may be considered a symptom of one form of the terminal affections of the heart due to cardio-sclerosis or old age. I have found it a precursor of steadily advancing cardiac weakness, and although for a time considerable relief may be afforded, the changes in the heart are so advanced that in the nature of things only one end can be looked for. In such cases pain may be absent.

§ 44. **Vagal sensory reflex.**—So far the symptoms I have dealt with have been mainly concerned with the reflexes connected with the sympathetic nerve supply. Equally instructive though less frequent symptoms can be shown to arise from stimulation of the vagus. At its centre in the medulla this nerve is in near relationship to the upper cervical nerves, and it would seem more particularly to the sensory nerves supplying the sterno-mastoid and trapezius muscles. Not only can these muscles become extremely hyperalgesic in various heart affections, but the pain from heart affection may be felt in the region of distribution of the cervical nerves. Pain may also be felt during an attack of angina pectoris in the gums and throat—

due to a radiation of the stimulus from the vagus to the centre of the fifth nerve.

Other very striking reflexes are sometimes met with in an attack of angina pectoris. Thus during or after an attack, an abundant flow of saliva and the secretion of large quantities of pale urine may occur ; both symptoms I suggest are due to reflex stimulation of the floor of the fourth ventricle.

There are also good grounds for attributing some forms of dyspnoea to reflex stimulation from the heart.

§ 45. **Conditions in which angina pectoris is induced.**—If a large number of cases be studied, symptoms of angina pectoris will be found to arise in patients with the most diverse forms of lesion, and even in patients without any evidence of cardiac disease. From among a number of conditions, I select the following : Cases of (1) aortic aneurysm, (2) aortic valvular disease, (3) atheroma of the coronary arteries, (4) myocardial degeneration or enfeeblement of the heart muscle from poor nourishment or over-exertion, (5) increased arterial pressure of the elderly (usually associated with 3 and 4).

It would seem that identical symptoms produced from conditions so diverse cannot be directly due to the organic lesion, to the aortic aneurysm, to the disease of the coronary artery, or to the increased peripheral resistance. We must therefore look for a cause common to all five conditions.

§ 46. **Conditions giving rise to attacks of angina pectoris.**—In order to find what this common cause may be, consideration of the conditions giving rise to an attack of angina pectoris will help materially. In the first place, it is to be noted that angina pectoris in the five conditions cited appears as a late symptom, after the heart has been struggling a long time against obstacles opposed to its efficient action, or after the nutrition of the muscle has been impaired by gross pathological changes in the coronary artery, or when the muscle-fibres have become impaired through slowly advancing degeneration of the fibres. We find, further, that many patients when at rest do not suffer from pain, but that any cause inducing increased work on the heart provokes an attack. Bodily exertion in any form, excitement, increased peripheral resistance (as in exposure to cold air), may bring on directly an attack of angina pectoris. That is to say, in predisposed individuals all circumstances that throw more work on the left ventricle induce an attack of angina pectoris.

§ 47. **Association of angina pectoris with exhaustion of the muscle of the heart.**—Such considerations lead to the conclusion that angina pectoris arises from certain conditions of the muscular substance of the heart, when the contraction meets with a resistance greater than it can easily and

efficiently overcome, whether a fairly strong muscle struggles against an increased resistance (as when there is great peripheral resistance or a narrowed aortic orifice), or when a weak or degenerated muscle has opposed to its contraction a normal or even a lowered pressure, but a pressure greater than the weakened muscle can readily overcome.

§ 48. **Association of angina pectoris with impairment of the function of contractility.**—If we inquire what function of the muscle-fibres is concerned in the production of angina pectoris, we shall find additional confirmation for the foregoing conclusion.

I shall later deal very fully with the fact that affections of certain functions of the muscle-fibres (Gaskell's functions [123]) can be demonstrated by means of graphic records of the cardiac movements. I have taken a large number of tracings from patients who have suffered from angina pectoris—during the attacks and when free from pain—and an analysis of these tracings enables me to say with confidence that angina pectoris can occur when the excitability, the conductivity, and the power to produce a rhythmical stimulus are unimpaired. There only remain now the functions of tonicity and contractility. The evidence of failure of the function of tonicity is mainly shown in the dilatation of the heart, and typical attacks of angina pectoris frequently occur in hearts perfectly normal in size. Therefore angina pectoris may occur without any evidence of the impairment of the function of tonicity. Seeing, then, that angina pectoris can occur in patients when four out of five functions of the heart muscle are demonstrably intact, we are led to inquire whether angina pectoris may not be due to an impairment of the remaining function, that is, contractility. When we come to look for facts that have a bearing on this question we find : (*a*) an *a priori* evidence in the fact that this is the function directly concerned in supplying the motive force to the circulation of the blood, and that it is the function that will necessarily become exhausted when an excessive resistance is opposed to the contraction of the heart muscle ; (*b*) the symptoms associated with and the conditions giving rise to the characteristic sign of depressed contractility (pulsus alternans), are of a similar nature to the symptoms in angina pectoris ; (*c*) angina pectoris may be associated with the pulsus alternans ; (*d*) in other hollow organs severe pains are evoked by contraction of the muscle-wall.

From this line of reasoning I would suggest that angina pectoris is an evidence of exhaustion of the function of contractility.

§ 49. **Summation of stimuli as a cause of angina pectoris.**—The fundamental functions of the heart muscle correspond to those of other involuntary muscles that form the walls of hollow organs ; these functions

being modified to suit its special work. Like the other viscera, the heart is insensitive when stimulated in a manner that provokes pain when applied to the tissues of the external body-wall. I may point out that a prolonged strong contraction of a hollow organ can produce pain, and that this is undoubtedly the cause of the severe pain associated with renal calculus, gall-stones, spasm of the bowel, and uterine contractions. Can the heart give rise to pain in a similar manner ? On account of the modification of its functions, the heart cannot pass into a prolonged state of contraction. Immediately it contracts, the function of contractility is abolished and the muscle passes at once into a state of relaxation, and for this reason the pain cannot be produced by a ‘ spasm of the heart ’. But I suggest that the heart muscle may produce pain when it is confronted with work greater than what it can readily overcome—a condition which produces strong peristalsis and pain in other hollow viscera. But the pain in the heart arises by a slightly different mechanism. A skeletal muscle will contract in obedience to stimulation of a sensory nerve going to the spinal centre of its nerve, if a stimulus of sufficient strength be applied. If the stimulus is too weak no contraction follows, but if this weak stimulus be frequently and rapidly repeated, then the muscle contracts in accordance with the law of the summation of stimuli. I suggest that the heart muscle induces pain on the principle of summation of stimuli. If we minutely study our cases we shall find that the pain rarely arises at the first exposure of the heart to the effort that induces the pain ; sometimes effort has been undertaken a few minutes before the pain comes on, and in certain cases it may not come on for hours after the causal exertion has ceased. From such observation we can infer that the heart muscle was exhausted by the exertion, and so great was the exhaustion of the reserve force that the heart was unable to regain its reserve with cessation of effort, so that the exhaustion persisted till it culminated in an attack of angina pectoris.

The reasonableness of this explanation will be more apparent after dealing specially with the symptoms of angina pectoris in individual cases.

CHAPTER VII

Angina Pectoris

In Chapter VI, I give the reasons for assuming that the symptom complex called Angina Pectoris belongs to the class of reflex protective phenomena where the symptoms are evoked by a viscus reflexly stimulating certain areas in the central nervous system. The stimulus from the heart to the spinal cord irritates the nerve-cells in close proximity to the nerve conveying the stimulus from the heart. The nerves thus irritated respond and exhibit the evidence of their peculiar function—sensory nerves, by pain felt in their peripheral distribution, motor nerves, by contraction of the muscles. In this way we get the peculiar distribution of pain in angina pectoris, and the sense of constriction of the chest-wall. This violent stimulation of the spinal cord may leave, after its subsidence, an irritable focus in the cord, rendering that portion of the cord more susceptible to stimulation, so that it becomes easier for future attacks of angina pectoris to be provoked. This irritable focus can be demonstrated to exist in some patients by the hyperalgesic state of the skin and muscles, and other subcutaneous tissues in the region where the pain was felt. So sensitive may this irritable focus become, that an attack of angina pectoris may be provoked by a stimulation reaching the focus from regions other than the heart. I have further suggested in the foregoing chapter that these phenomena are not the outcome of the gross lesion found in cases of angina pectoris, but are due to the exhaustion of the contractile function of the heart muscle. If this view be kept in mind, the examination of patients is greatly facilitated, and the grounds for a rational diagnosis, prognosis, and treatment are laid.

§ 50. Conditions predisposing to an attack.—That a muscle should

evoke disagreeable symptoms when over-fatigued is a principle applicable to all muscular structures of the body. The peculiar functional attributes of the heart muscle imply a modified method of exhibiting these disagreeable symptoms, which I have dealt with in Chapter VI, showing how in many cases it is probably due to the law of summation of stimuli. In looking at the coronary arteries in certain typical cases of angina pectoris, one can reasonably infer one way in which the attacks are brought about. In some cases the coronary arteries are so narrowed as scarce to permit the entrance of a pin. During life the stream of blood must have been greatly reduced, and if it were sufficient to supply the muscle during rest, it was demonstrably insufficient while the heart was in a state of activity. In this respect there seems to be a distinct affinity between the origin of the pain in these cases, and in those cases of what is called 'intermittent claudication'—first described in the horse, but not infrequent in man. In one of my cases of this complaint, for two years the patient could not walk one hundred yards before he had to stand still and rest on account of the great pain in the legs and feet, and of intense coldness of his feet. On standing a few minutes the pain disappeared, and his feet became warmer. Here there was a marked atheromatous degeneration of the arteries of the leg, and the supply of blood was sufficient for the muscles when at rest, but contraction of the muscles demanded a greater supply than the arteries could furnish, with the result that the exhausted muscles became painful, and the diminished supply of blood to the skin caused a sense of coldness. Ultimately the arterial supply became so slight that gangrene of the toes set in. From the foregoing illustrations a fairly safe conclusion can be drawn as to the manner in which anginal symptoms are provoked. It can further be safely inferred that the muscle exhaustion may arise in other ways, for example, when the nutrition is poor, as happens in anaemic and badly nourished people. The heart may become exhausted because it is never allowed sufficient time to recover its reserve force, as in individuals who are worried and overworked. Or exhaustion may arise from obstruction to the work of the left ventricle, as in disease of the aortic valves, and in atheroma of the arteries, especially when accompanied with partial obliteration of the capillary field (§ 221). In these cases the disease that has damaged the aortic valves and arteries has at the same time invaded and enfeebled the heart.

In addition to exhaustion of the heart muscle as a cause of angina pectoris, there has to be noted this further fact, that angina pectoris arises only in connexion with exhaustion of the left ventricle. The symptoms of angina pectoris are invariably associated with lesions on the ventricular side of the

mitral orifice. In a few cases I have found angina pectoris in patients with mitral stenosis after violent exertion, but it is to be noted that the blood-supply to the ventricle is limited in such cases. I do not remember having seen a case of angina pectoris with free mitral regurgitation unaccompanied by affections of the aortic valves; hyperalgesia of the chest-wall is, however, fairly common when there is also dilatation of the heart. In several instances I have noted, with Broadbent and Musser, the complete cessation of anginal attacks with the onset of free mitral regurgitation. In such cases the attacks were due to the left ventricle struggling to overcome the high arterial pressure, and the cessation was due to the dilatation of the heart rendering incompetent the mitral valves, and thus permitting the ventricle to ease its load (Chapter XXVII). Though freedom from attacks of angina pectoris was thus acquired, a great fall of arterial pressure resulted, leading to dropsy and gradual exhaustion (Chapter XXII).

§ 51. **Conditions inducing an attack.**—Angina pectoris is never the outcome of an acute affection; it is invariably led up to by a long period of gradual exhaustion.* The onset is, in the majority of cases, induced by some extra effort on the part of the heart. This need not be an effort of unusual severity; it occurs more frequently when the individual is using his powers in a way which formerly required no very marked strain. The cause may be of the nature of bodily exertion, mental excitement, exposure to cold air, or anything that calls for more work on the part of the heart. In most cases the attack does not come on at once, unless the effort is persisted in, as in walking up a hill, and the first warnings of heart exhaustion are ignored. The attack of pain may not come for some minutes or even hours after the causal exertion has ceased, and when the patient has been resting in bed. In some severe cases the attack of pain may come on at intervals when the patient has been exposed to no exertion, particularly in cases where the attacks have been frequent, and a tendency to them has been established.

§ 52. **Character and duration of an attack.**—The attacks may be so slight at first as to pass almost unnoticed, and it is only after they become more frequent and severe that the patient's attention is called to the fact that he had previously experienced some discomfort. In the more simple cases the pain may not be very severe, and, appearing after some violent exertion, may never again show itself. Instead of pain it may be but a slight sensation of constriction across the chest, that calls for a deep inspiration

* Except in cases of coronary embolism, of which there are several cases on record in which attacks of angina pectoris were evidently due to the plugging of a coronary artery (Garrod[6] and Parkes Weber[55]).

to relieve the tension. The more severe attacks imperatively command a cessation of all efforts, and here all degrees of suffering may be experienced. During the attack the patient may stand still in a position of rigid immobility, afraid to move or to speak, scarcely daring to breathe, or he may kneel down and rest his head on a chair, or roll on the floor in an extremity of agony, or the patient may become unconscious and, in rare cases, may die. If the pain is located in the arm, he may nurse it across his chest, rocking backwards and forwards. The face may become pale or flushed, and beads of perspiration may roll down the forehead. The attack may terminate with the expulsion of air from the stomach, unconsciously sucked into the stomach during the attack (§ 64).

In most cases the attack lasts for a few seconds, but it may continue with great severity for several hours, yielding only to large doses of opium. Such severe cases may die during the suffering.

§ 53. **The symptoms present during an attack of angina pectoris.**—The chief symptom is pain : there may be also a sense of constriction across the chest, a sense of suffocation, and a sense of impending death. Occasionally other reflexes may be present, such as a flow of saliva from the mouth, and an increased secretion of urine. These symptoms are not all present in every attack, nor are they always present in the same degree. There may be a slight pain or a slight sense of constriction across the chest. Or the pain may be of the utmost severity, with constriction of the chest so violent that the patient feels as if his breast bone was about to break.

Pain.—Pain is usually referred to some portion of the distribution of the upper four left dorsal nerves in the chest and arm. Sometimes the pain may be felt as low as the distribution of the sixth dorsal nerves in the epigastrium, and as high as the eighth and seventh cervical in the ulnar border of the forearm and hand. It is rarely felt in similar areas on the right side, and sometimes it is felt in the neck and back of the head, in the upper cervical nerves, whose roots are in close association with the vagus. The pain is usually felt across the chest, and may remain stationary there, or it may radiate in a very definite manner into the axilla, and down the arm to the ulnar border of the forearm and hand. When it does this it may stop for a brief period in the upper arm or forearm, and be felt there most violently. On the other hand, the pain may start in the arm and radiate to the chest, where it remains with great severity for a time.

Constriction of the Chest.—Arising along with the pain, or following it, or quite independent of the sensation of pain, is a sense of constriction, which I have reasoned is due to the reflex stimulation of the intercostal muscles. It may be so slight as to be felt only as a mere tightness across the chest

following exertion, or it may grip the chest so firmly that the patient has to stand still and take a great deep inspiration to relieve the spasm of the muscles. In its most violent form it adds greatly to the suffering of the patient when pain is also present. Thus a man aged forty-eight, who had violently exerted himself, felt a pain in his chest come on gradually some minutes after the exertion. As the pain increased he called on me, and I asked him if he felt his chest constricted. He said ' No '. A few hours later, the pain increased in severity; then suddenly he felt his chest gripped with a violence so great that it added, he said, indescribably to the terror of his suffering. It was only relieved by large doses of opium. Next day he felt for a short time when in bed as though that ' awful gripping were coming on ', and he lay for ten minutes with perspiration pouring off him, in the dread of its return.

Feeling of impending dissolution.—This, I presume, is the result of violent stimulation of the nervous system comparable to what happens when any other viscus is violently stimulated, as after a blow in the epigastrium, or on the testicle. On rare occasions the patient faints during an attack, and in one instance a patient of mine never recovered, but died during the faint.

§ 54. **The state of the heart and arteries.**—The observations of Nothnagel[39] that contraction of the arterioles may precede an attack of angina pectoris, and of Lauder Brunton[4] that the peripheral arteries may contract during an attack of angina pectoris, have led to some misapprehension regarding the circulation in this condition. I have had the opportunity of examining a large number of patients during attacks of angina pectoris, in some of whom the onset occurred immediately after exertion, in others some hours after exertion; in others the attacks have come on suddenly when the patient was in bed. I have taken a large number of sphygmographic tracings and a few blood-pressure observations during these attacks, and have never found a single case where the arteries were constricted in the manner sometimes described (see illustrative cases in Appendix I). In six cases where the patient was seized with the pain in bed, and died during the attack. there was not the slightest sign of contracted arteries. In one of these cases I measured the blood-pressure during an attack shortly before his death. and found that the pressure had fallen considerably. I have seen and carefully studied a few cases of Nothnagel's ' vaso-motor angina pectoris ', and can positively state that they come under a different category from the more common forms of angina pectoris due to disease of the coronary arteries (§ 66). I can only infer that cases of arterial spasm are very exceptional, and their description, fostered by the relief obtained by the adminis-

tration of amylnitrite, has given quite a wrong conception in regard to the conditions inducing an attack of angina pectoris. I have found during these attacks the pulse became small, soft, and scarcely perceptible, from weakness of the heart, the heart sounds becoming very faint. I have also found acceleration of the heart's rate—in one case very considerable,—and occasionally the occurrence of an extra-systole, followed sometimes by a characteristic pulsus alternans. In a few cases irregularities have occurred whose nature I have not been able to determine satisfactorily, but in the vast majority of cases I could detect no change in the heart or arteries, and there never was the slightest enlargement of the heart coming on during the attack.

§ 55. **Symptoms after an attack.**—The patient feels greatly exhausted after the attack has passed off. Sometimes the pain does not completely disappear, and an uneasy painful sensation may last. The end of an attack may coincide with the expulsion of air from the stomach, and as this is usually accompanied by a sense of relief, the attacks are often supposed to be of gastric origin. I have watched these patients, and have no doubt that the air has been sucked into the stomach during the attack. This air suction is seen most characteristically in nervous people, especially women, and is referred to more fully in § 64. Some patients have a desire to micturate, and the urine secreted is always abundant, pale, and of low specific gravity. In some patients areas of hyperalgesia of the skin appear in some portion of the field in which the pain is felt. In the first attacks it may be limited to a small patch, as in Fig. 11, but with recurring attacks the area may spread wherever the pain is felt, as in Fig. 12.

§ 56. **Establishment of a tendency to recurrence of the attacks.**—I have already described how an irritable focus may be produced in the cord after a violent stimulation from a visceral lesion (shaded area in Fig. 8, p. 36). This may be manifested by an area of hyperalgesia in some part of the body, and in those that exhibit this hyperalgesia attacks of angina pectoris come on with very little provocation. Even when there is no distinct evidence of hyperalgesia, the stimulation of the skin or the movements of the muscles of the arm may induce an attack. I have on several occasions inadvertently brought on an attack while testing the sensibility of the skin and deeper tissues over the praecordium. Visiting a patient one day, who suffered from violent attacks, I found him feeding himself entirely with his right hand. When I asked him why he did not use his left, he said he was afraid to do so, as sometimes the movement of the left arm induced an attack. He died a few hours after, during an attack.

§ 57. **Prognosis.**—The tragic circumstances surrounding certain cases

of angina pectoris have so oppressed the profession and the laity that an altogether exaggerated opinion has been formed of the gravity of this complaint. If it be realized that angina pectoris is but the expression of an exhausted muscle, and that the exhaustion may arise from any cause that

Fig. 11. The shaded area shows the position of a patch of cutaneous hyperalgesia after the first attack of angina pectoris.

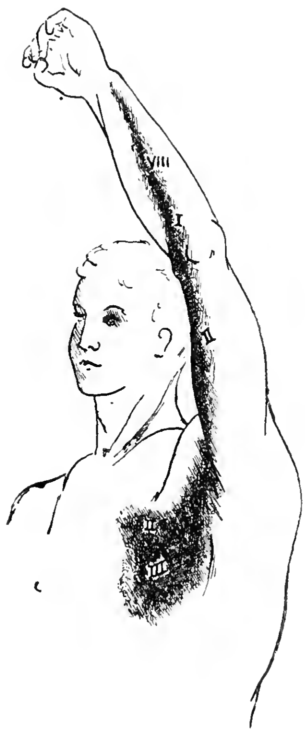

Fig. 12. The shaded area shows the distribution of the pain and cutaneous hyperalgesia after repeated attacks of angina pectoris. From the same patient as Fig. 11 (compare the shaded area with the distribution of the eruption in Fig. 10). The roman numbers refer to the nerves implicated, viz. I, II, and III—the first, second, and third dorsal nerves, and VIII, the eighth cervical nerve.

overtaxes the heart, a truer appreciation of the meaning of the symptoms will be obtained. The estimation of the gravity of the cases does not depend upon the violence of the symptoms. A severe attack is not necessarily serious, nor is the mildest free from danger. The importance of the symptoms must be estimated by an examination of the conditions that have induced the muscular exhaustion. This is, as a rule, not a matter of

much difficulty, if one carefully searches for a predisposing cause. The age of the patient, the conditions of his or her life, work, worry, nourishment, over-indulgence in tobacco, and, in the case of women, the number of children she has borne, her menstrual functions, all lead to the recognition of the nature of the muscular exhaustion. If the probabilities point to the absence of progressive arterial degeneration and degeneration of the heart muscle, the patient may on the whole be assumed to have a favourable future. If one has reason to suspect that the symptoms have appeared in the early days of cardio-sclerosis, when the patient, ignoring the limitation of his powers, follows the manner of his life pursued in vigorous manhood, then the probabilities are that, with rest and care, the heart will recover from its exhaustion, and be able to carry on its work for many years in comfort. Where the prognosis is most serious is when it occurs with marked evidence of general arterial degeneration, and where there is little response to treatment, and where the attacks are induced with slight provocation. These are the circumstances that would lead one to look gravely upon this condition. But even when cases are so severe that for months a patient may scarcely be able to walk across the floor without inducing an attack, rest for a long period may restore the heart and induce a cessation of the symptoms ; otherwise the condition arrived at is practically beyond the possibility of recovery.

§ **58. Treatment.**—Treatment naturally divides itself into two heads, namely, the improvement of the condition of the heart, and the giving of relief during an attack.

Improvement of the condition of the heart.—For the first of these it must be borne in mind that the attack is the expression of an exhausted muscle, and the treatment requires a careful inquiry into the conditions inducing that exhaustion. First and foremost, a stop must be put to that form of exertion which has induced the attack, and any other conditions that predispose to it must be avoided, such as work, worry, sleeplessness, over-indulgence in food and alcohol, tobacco, and so forth. The next step is to place the heart in such a position that it will regain its reserve force, that is, the heart must be given less to do. This does not necessarily mean that the patient should rest in bed, but he should restrict his movements as much as possible. Here the habits and condition of the patient will have to be considered, and each individual separately treated. To stop an individual altogether from his work and engagements may be most serious. It is best, as a rule, to permit him to follow some particular kind of work that does not put a strain upon his heart. When feasible, a complete change of habits and life, as free from over-exertion as

possible, is most beneficial, such as a good holiday spent in the manner that affords the maximum of enjoyment and requires the minimum of effort. In the cases that do not yield to such limited restriction, and when the attacks are demonstrably the outcome of an advanced exhaustion of the heart, then absolute rest in bed is necessary. In other conditions the treatment should follow the lines that I have laid down in the chapters upon treatment. Of remedies and methods supposed to cure, the name is legion. Happily in their prescription the above suggestions are also included, and the benefit so accruing is too often attributed to the remedy or method. I have in several individual patients tried various so-called remedies, and I find on the whole, that if the patient is not worried, has plenty of sleep, and leads a fairly restful life, he does as well without any special drug treatment as he did with it.

As many patients of a neurotic type suffer from angina pectoris the mental factor should always be considered. The term angina pectoris conveys to their minds such fearful associations, that they readily become depressed and miserable. A careful study of each case will show that in the majority the attack is the outcome of a temporarily exhausted muscle and the patient can have his mind relieved by the assurance that the prognosis is a good one. In the after-treatment of such cases careful management is needed, in order that they should not always be reminded of their complaint. Hence systems of dieting, where the patient at each meal has to reflect if the ingredients are injurious to his heart, should be rigidly avoided. Most dietetic systems are the outcome of a fad, and based on an imperfect knowledge of digestive and metabolic processes, as in the exclusion of common salt and lime salts from the meals. For the same reason ‘ health resorts ’ where people congregate and discuss their ailments should be avoided.

Treatment during an attack.—The slighter attacks require no treatment. When they become more severe, rapidly acting vasodilators should be administered, such as hot drinks, hot water with whisky or brandy, and, best of all and most speedy, amylnitrite by inhalation. This drug is not successful in all cases, but in many its action is rapid, and the relief is generally complete. When it is successful, it has been inferred that the patient had previously constricted arterioles, or increased arterial pressure, and that the pressure was reduced, and so the heart was eased. This is not the full explanation. A patient with cardio-sclerosis had an attack of angina pectoris in my consulting-room. I took his blood-pressure, and found it 190 mm. Hg., and then administered to him nitrite of amyl : it acted instantaneously, and gave him perfect relief. After fifteen minutes I again took his blood-pressure, and found that it had risen to 200 mm. Hg.

Though the pressure was higher, he had no pain. I inferred that the action was similar to that of a man who puts on a hat that is too tight ; at first there is no pain, but gradually, by the summation of stimuli, discomfort comes on and increases in intensity ; he removes the hat from his head, and obtains relief. He replaces the hat in the same position, and, although it is as tight as before, the pain has permanently disappeared : so that the temporary removal of the summating stimuli seems to be the reason for the relief afforded in the particular case mentioned.

When nitrite of amyl fails to relieve the patient, we are forced to use chloral or morphia in doses sufficient to give relief. I have found occasionally that chloral acts beneficially not only in relieving the somewhat long attacks, but in preventing the attacks recurring, when given in repeated small doses of 3–5 grains and also when given at night to induce sound sleep.

In advanced cases where the suffering comes on mainly at night, it is occasionally very difficult to give the patient relief. Massive doses of oxygen have in some cases, as in cardiac asthma, been followed by marked benefit, and should be tried (see p. 279).

CHAPTER VIII

Heart Affections and a Hypersensitive Nervous System

It not infrequently happens that the most common forms of disease are the most difficult to describe. I attempt here the analysis and explanation of the symptoms present in certain cases which are frequently met in actual practice. As the symptoms present great variety in number and intensity, numerous attempts have been made to divide them into groups, and we find them under various guises, as neurotic hearts, cardiac neuroses, cardiac neurasthenia, pseudo-angina pectoris. I have endeavoured to find the underlying principles which provoke these manifold symptoms, as their due appreciation is of prime importance in the management of these cases.

§ 59. Reaction of visceral disease on the central nervous system.—In describing the symptoms of angina pectoris (Chapters VI and VII), I have endeavoured to show that the symptoms arise from a reflex stimulation of the central nervous system. But the heart and the nervous system can react upon one another in other ways than by reflex stimulation. In heart affections, as in affections of all other viscera, there is a tendency for the central nervous system to become hypersensitive (I use this word for want of a better) whereby symptoms of nervous origin are readily evoked. This applies more particularly to the production of sensory phenomena, where a comparatively small visceral lesion gives rise to an irritable focus in the spinal cord, and to extensive suffering and widespread areas of hyperalgesia, or to certain mental states where the patients sometimes become 'nervous' and apprehensive. The result of the association of heart affections with

these latter, may be summed up in the expression that the cardiopath tends to become a neuropath.

This mental state is seen very characteristically in both men and women. If they have been told that they have a murmur, or an irregular heart, or if they are conscious of an extra-systole, or suffer actual distress of a cardiac origin, they become extremely apprehensive. The hyperalgesia, so common in the breasts of women suffering from some slight heart trouble, is a constant source of worry, and some are continually imagining that the abnormal soreness is an indication of serious disease. This apprehension is unfortunately too often aggravated by the warnings of the physician, who estimates the significance of the symptoms too seriously, or will neither admit nor deny the gravity of the condition.

The combination of cardiac and nervous exhaustion may be brought about in another way. People with a tendency to 'nervous debility', or who have acquired it from some other cause, may develop some cardiac trouble, functional or organic. In such people, the reflex symptoms are greatly exaggerated. Thus one of my patients with aortic and mitral disease experienced no sensory phenomena until she developed a gastric ulcer. This gave rise to great pain, and to a widespread area of hyperalgesia of the skin and muscles in the left side of the abdomen. Soon after this she began to suffer from pain from the heart affection, and the hyperalgesia finally embraced nearly the whole of the left chest. The patient lived for many years after the appearance of these symptoms, and a pyloric ulcer and aortic and mitral valve disease were found on post-mortem examination.

In all these cases we must exercise a great deal of judgement. It frequently happens that in patients with a demonstrable cardiac lesion, the symptoms are estimated too seriously, and the case looked upon with greater gravity than need be. On the other hand, if there be no murmur or irregularity, the case is liable to be treated lightly as one of 'pseudo-angina pectoris' or neurasthenia.

§ 60. **Pseudo-angina pectoris, a useless and misleading term.**—It is time the term 'pseudo-angina pectoris' was dropped out of medical literature. While it may be convenient to group under indefinite terms many conditions of whose nature we are ignorant, it should be borne in mind that this grouping is but provisional, and a confession of our ignorance of the real nature of the complaint. With advance in our knowledge, first one complaint and then another should be placed in a group whose cause is definite and known. In this way many cardiac terms, such as tachycardia, embryocardia, bradycardia, have been employed loosely, and now

should never be employed unless a definition be given of what is meant.

The term ‘ angina pectoris ’ is employed to designate a group of symptoms evoked by the heart, of which pain is the most distinctive. As angina pectoris is sometimes associated with grave organic lesions, we find such cases referred to as ‘ angina pectoris vera ’. The term ‘ pseudo-angina pectoris ’ is applied to cases in which the pain resembles that of angina pectoris vera, but is due to some other cause than heart disease, or in which the pain arises from the heart with no organic lesion. In regard to the former class, if the pain is due to some other viscus, e.g. the stomach, as sometimes happens, why call it ‘ pseudo-angina pectoris ’ ? If it is due to the stomach, why not say so ? In regard to the latter class, the employment of this term is due to a total misconception of the nature and mechanism of visceral pain. The fundamental cause of the pain is the same in the case of the heart as in that of any other viscus, and the pain is as readily induced in the heart as in the stomach. As we would never dream of calling a stomach pain a ‘ pseudo-gastralgia ’, so we need not call a heart pain ‘ pseudo-angina ’.

I deal with the matter at length, as the employment of a fine-sounding term has too often sufficed for a diagnosis, so that no inquiry into the real nature of these symptoms has been undertaken. In the great majority of cases when a patient complains of a pain in his chest which radiates into his arm, in the area shaded in Fig. 12 (p. 51), the pain is of cardiac origin. The only other conditions in which I have found pain to occupy this characteristic site, was in herpes zoster affecting the upper dorsal nerves and certain rare forms of gastric spasm. In one case, I thought I had to deal with a case of angina pectoris until the herpetic eruption revealed the true nature of the complaint. It is quite conceivable that other conditions may give rise to pain having this distribution, but that is no reason for calling them pseudo-angina pectoris.

The characteristic distribution of the pain and other sensory phenomena at once excludes hysteria, for in the latter the symptoms do not follow the anatomical distribution of nerves. When a hysterical patient feels a pain in this region, it may be assumed that there is probably some cardiac trouble in addition to the hysteria.

§ 61. Exaggerated sensory phenomena with and without valvular disease.—A great many people with a demonstrable cardiac lesion, as of the aortic or mitral valves, develop sensory phenomena in an exaggerated form. This is particularly seen in some women in whom the reserve force is exhausted. Such folks may struggle on for a long time, working hard and ignoring their earlier symptoms of a limitation of the field of cardiac

response, determined not to give in. Finally the nervous system shares in the exhaustion, and the breakdown is brought about with an extreme development of the sensory phenomena ; thus attacks of pain, sometimes of great severity, may be felt across the chest and extending into the left arm, or more often the complaint may be of a dull aching pain of varying severity, but distinctly worse at the end of a day's work. The hyperalgesia may spread over a very extensive area, and sometimes is extremely acute.

On account of the manifest lesion in the heart these cases are not unfrequently diagnosed as cases of angina pectoris of a severe and dangerous form, and I have known them lead a life of great restriction for many years under this mistaken notion. The attacks are indeed those of angina pectoris, but are not dangerous, and are an evidence of exhausted heart muscle, and disappear with the restoration of the reserve force.

On the other hand, we have mothers of families with no heart murmurs, who for many years have worked hard from morning till night, whose sleep has been disturbed by ailing or fretful children, and who finally break down with exhausted heart and nervous system. Some of my most typical cases have been in young women whose sleep has been disturbed frequently every night for many years to attend to an ailing parent. This constant strain night and day exhausts the strength. These patients suffer from heart pain, sometimes with the classical symptoms of angina pectoris described in Chapter VII.

With suitable management they eventually recover, though recovery is usually very protracted, the patients sometimes having to lead very quiet lives for months or even years. Similar symptoms may arise in others who have been exposed to worry and anxiety, or who have suffered from sleeplessness, while others may suffer when there is no apparent reason for the exhaustion.

§ **62. Exaggerated sensory phenomena in early cardio-sclerosis.**— The possibility that in these cases there may be beginning cardio-sclerosis, should be kept in mind, especially in cases over 40 years of age. There is nothing to tell whether it is so or not, for the superficial arteries may be quite normal in appearance, and the blood-pressure give no sure information. This needs to be specially insisted on when these exaggerated phenomena appear in women between 50 and 60. I have seen a number of patients develop all the sensory phenomena in an extremely exaggerated form, becoming weaker until they were unable to leave their beds ; some have become unconscious, and died ; others regained consciousness, and after a time sufficient strength to go about for years. After their recovery I have been surprised to find an aortic systolic murmur, which had not been present

prior to the breakdown. Some of these have remained liable to attacks of angina ; one dropped down dead, and in another, who died from subsequent heart failure, there was marked sclerosis of the heart muscle, coronary arteries, aortic valves, and aorta. The diagnosis in these cases depends on the response to treatment (§ 57).

§ 63. **Characteristics of the sensory phenomena.**—There are some special points in these cases that distinguish them from those who do not have the same susceptible nervous system. The suffering may not be as severe as in the more grave forms of angina pectoris, but it is more lasting and comes on after periods of continuous exertion. Sometimes it is limited to the left arm, if that arm has been much employed in work, as in washing or baking. It is frequently associated with extreme tenderness on pressure of the tissues of the left chest and neck, especially the left breast, the pectoralis major and sterno-mastoid muscles. After testing the tender skin and muscles by slightly pinching the skin and muscles between the fingers and thumb, the part becomes extremely sore, and the aching lasts for hours afterwards. When the patient is suffering from severe pain, the mouth may become dry and parched, and large quantities of pale urine may be passed, as happens in cases where the angina pectoris is of very grave significance.

§ 64. **Air suction.**—Another symptom is extremely common in these cases—the belching of air. One searches textbooks in vain for any hint as to the nature of this symptom, and though it is extremely common in all neurotic people, its significance is almost invariably misunderstood. A detailed and satisfactory account is given by Wyllie,[441] and it is to this article I owe enlightenment as to the meaning of this symptom.

The chief feature is the noisy expulsion of air from the stomach. Patients complain of attacks of flatulence, and in these attacks seemingly expel large quantities of air, but if closely watched they will be seen first to suck air into their stomachs. Before expelling the air, they unconsciously close the glottis, fix the muscles of the abdominal wall, then expand the chest. As no air goes into the lungs, and the diaphragm is raised, the pressure in the stomach becomes negative. By this process they suck air into the stomach. After sucking in a quantity, they expel it with considerable force, and often with a good deal of noise. Many people can do this at will, others only in certain states of excitement. Some have 'attacks of flatulence' in the middle of the night, and such attacks are due to air swallowing, or more correctly air suction. As Wyllie points out, these attacks can be stopped by making the patient open his mouth widely, and keeping the jaw propped open by a large cork between the teeth, a procedure which prevents the air suction.

I have watched several patients during an attack of angina pectoris, and when they stand seemingly immobile, they unconsciously suck air into the stomach. Immediately the pain subsides the air is expelled, and the patient is apt to attribute the relief he has experienced to the coincident and demonstrable act. This very obvious phenomenon has led many observers to imagine that the attack was gastric in origin, and hence the group of gastric 'pseudo-anginas'.

This association of air suction with attacks of angina pectoris, which is sometimes found in men, is extremely common in women. As air suction is frequent in women, it is sometimes mistaken for a hysterical symptom, and its relationship to a real heart attack is apt to be overlooked. As a matter of fact, attacks of air suction are apt to arise from any exciting cause, and attacks of angina pectoris readily induce them. In some it occurs so readily that it may come on before the real suffering. Thus, one lady who suffered from extreme arterial degeneration, had severe attacks of angina pectoris, which disappeared after a long period of rest in bed. When she got about again, she could walk on the level with comfort, but the slightest hill brought on discomfort, which if not heeded resulted in great pain in the chest. As a rule, however, before the pain became severe she began to suck in and to expel air. She would rest a minute and start again, but soon had to stop, and expel 'more wind' as she put it.

§ 65. **The circulatory symptoms in the X disease.***—There is another class of cases of somewhat indefinite character, that needs to be recognized in order to appreciate other forms of heart trouble. The class I allude to will be recognized by every practitioner as they form a considerable portion of the community. The individual is spare and thin; the face is often drawn and lined, sometimes even in the young. It is usually pale, though in some the face is ruddy, and the nose is red in cold weather. The hands are usually cold, and they tell you their circulation is feeble. They are always worse on raw cold days, and feel chilly and ill after a cold bath. Their complaints are extremely varied, and many have a fixed idea that certain organs are at fault, and it is true that some trouble, usually slight, may be found in some organ. Thus we find gastric and bowel complaints extremely common, though other viscera may also be at fault and com-

* I employ the term 'X disease' for the reason that I do not know the nature of this complaint. Many physicians call members of this class 'neurasthenics', and are content to leave the matter there. This is simply to give a complaint a name, which is so satisfying that the fact is often lost sight of that the name sheds no light upon the complaint and is nothing but a cloak for ignorance. If the term 'X disease' be employed it will be a glaring acknowledgement of our ignorance, and will lead to a constant endeavour to clear up the mystery surrounding these cases.

plained of. The patient's mental condition is curious and interesting. Some of them are sane, level-headed, and extremely intelligent. To these the bodily suffering is nothing more than a grievous and troublesome affliction. In others it leads to irritability and peevishness in temper. Some become introspective, and are deeply concerned about their bodily or spiritual affairs. It alters their views of material things; cranks and faddists, political, religious, and dietetic, are common among them, often exhibiting strenuous enthusiasm for their particular ideas. Another astonishing feature in these cases is the remarkable way in which a temporary recovery may take place. For weeks some of these folks may go about miserable and ill, taking little food, finding that little too much for the digestion, and searching for some kind that will suit them—when suddenly they feel better. Their recovery may last for weeks or even months, but they generally relapse.

Now this peculiarity leads to another characteristic of this complaint—unbounded and unreasoning belief in what they take to be the cause of their recovery, diet, drug, methods of exercise, operation. It is because of this tendency to recover that there are so many cures. If one reads between the lines of the testimonials in favour of certain remedies, empiric or recognized by authority, we can see that it is this class of case that is being treated. It is especially among them that faith-cures abound, and these are the people who swell the ranks of Christian Scientists. Emotional excitement, whether of love or religion, always relieves this kind of person, and so when religion comes into play we get the various forms of faith-healing. Many women feel extremely well when pregnant.

The diagnoses of medical men are as numerous and varied as the complaints of the patient. The gynaecologist diagnoses some pelvic disorder; the surgeon sees the source of all the trouble in an appendix, a dilated stomach, or a wandering kidney; while the physician recognizes the disease according to the bent of his studies—a heart affection, visceral stasis, gastroptosis, neurasthenia, atonic dyspepsia, and so forth. So minute indeed are some of the diagnoses, that we find them classified further as cardiac, gastric, mental, or renal neurasthenias.

I enter into the description of this class of case somewhat fully because many of them masquerade as cases of heart disease. I have for some years been inquiring into the nature of the symptoms in these cases, and here detail some of the cardio-vascular phenomena, as the lack of the recognition of the nature of these symptoms often leads to a mistaken diagnosis.

The most outstanding feature is cold hands, and this is sometimes associated with a peculiar roughness and thickness of the skin. The fingers may

become white and numb ; 'dead' is the term often applied. Exposure on a very cold day may cause the condition to be so extreme that pain in the finger ends is very severe, and in one case I have seen a slight gangrene follow. The nose is often red, and the association between dyspepsia and the red nose is extremely common in these folks. There is very often dilatation of the stomach, associated with accumulation of blood in the abdomina veins. This latter can be demonstrated in several interesting ways. If the patient be laid on his back, and very gentle pressure be steadily applied with the hands placed on the upper part of the abdomen, so as not to interfere with the respiration, the veins of the neck will gradually be seen to swell and the pulsation in them becomes greatly increased. In some cases this may be seen to occur during quiet respiration, the swelling of the vein occurring during inspiration. The cause of the swelling in this condition is that pressure on the abdomen empties the abdominal veins into the right heart, so that there is less accommodation for the blood returning by the superior vena cava ; hence the jugular vein distends. Inspiration causing a descent of the diaphragm compresses the abdominal contents, including the large veins, against the unyielding wall, and brings about the same result. G. Oliver says that by putting a bag of shot (14 lb.) on the abdomen in these cases he can raise the pressure in the arterial system.

The heart itself in these cases is sometimes slightly dilated, and there may be mitral and tricuspid systolic murmurs. They are very evanescent, present at one minute and gone the next. Sometimes we can detect them at the beginning of an examination, and in a few minutes they have disappeared. The rate and rhythm of the heart often varies. Sometimes it is rather slow, and sometimes it is irregular, the irregularity usually being respiratory, though occasionally extra-systoles are present, and then the patient if conscious of them is often greatly frightened, particularly if the doctor does not convincingly reassure him. Hesitation or doubt on the part of the doctor hangs like a cloud over the patient.

I have been particularly struck with the slow respiration in a number of these cases. It may fall as low as seven per minute (Fig. 77, Plate II), and the patient be free from any distress, and quite unconscious that anything is wrong. It is then that the heart rhythm is most affected, and the swelling of the vein during inspiration and from pressure on the abdomen occurs most characteristically. The nature of this irregularity is fully described in Chapter XVIII. A healthy individual can sometimes produce this irregularity by simply breathing slowly and deeply at the rate of seven or eight per minute.

§ 66. **Vaso-motor angina pectoris.**—For many years I was at a loss

to understand the cases described by Nothnagel[39] as vaso-motor angina pectoris. The term is such a fine-sounding one that it soon found its way into current literature, but I could never find it employed in the sense in which Nothnagel used it, nor did the cases recorded by others correspond with those given by him. I have come across several patients who suffered from angina pectoris, and a careful scrutiny of their symptoms leads me to think that these are the kind of patient described by Nothnagel. They are particularly liable to cold and chilly attacks. Occasionally the attacks of chilliness persist, and, if the patients have no opportunity to warm themselves, increase in intensity, until after an hour or two of misery they culminate in attacks of pain in the chest, radiating into the arm. The pain in severe cases persists with varying intensity until the patient gets thoroughly warmed.

I deal particularly with this subject because the term ‘ vaso-motor angina pectoris ’ conveys to many minds some definite idea of what happens in these cases, and though I can formulate no explanation that would, to my mind, satisfactorily explain the matter, I may point out that in these cases, the blood-pressure is not increased as one would expect, if the explanation were merely that the heart is acting against increased peripheral resistance. Although I have not taken the blood-pressure during a severe attack of pain, I have taken it in a number of cases when the extremities were cold and chilly and the patient suffering considerable distress, and the pressure has invariably been low—about 120–130 mm. Hg.

§ 67. **Prognosis in cases with exaggerated sensory symptoms.**—If care be taken to differentiate between the patients who exhibit exaggerated nervous phenomena due to progressive organic lesions, and those due to exhaustion apart from a progressive lesion, the prognosis can be made with fair certainty. Recovery almost invariably results in the latter class, though it may be delayed for a long time. Naturally, the complication producing the nerve exhaustion must be taken into account, and if due to other visceral affections, the prognosis depends also upon their nature.

When there is an organic lesion, as cardio-sclerosis, or valvular disease, on the whole the exaggerated nervous phenomena do not add to the gravity, but, it has even seemed to me, act favourably in many cases, for the early exhaustion is attended by such an amount of suffering, that the heart is protected from more extensive exhaustion of its reserve force.

In the cases included in the groups of X disease, even when angina pectoris is present, I have never found the heart trouble give rise to a serious breakdown, nor have I ever found death from heart failure in this type of patient. In fact, they are among the long-lived, for they usually

take such care of themselves that they avoid all risks and exposure to danger.

§ **68. Treatment.**—It is of the first importance that we should appreciate the nature of the trouble in these cases, and bear in mind the part played by the nervous system. The nervous element is often the chief one to be considered, and the sufferings of many of these patients are aggravated by the consciousness or dread of some serious affection of the heart. Having satisfied ourselves as to the real nature of the trouble, we should first of all reassure the patient. In a great many cases success in treatment depends on this, and we can often see patients at once made well, or greatly improved, when they become fully reassured. This is more particularly the case when the patient has previously been alarmed by being told that the condition was serious. The peculiar mental factor that makes this class of patient the stay and support of many forms of empirical or semi-empirical treatment, should be kept in mind. As suggestion plays an important part in the numerous special methods of cure, it should be used intelligently by medical men, and in a legitimate manner, that is to say, the patient should be reassured from the standpoint of a full knowledge of his condition.

There is a great tendency for the physician to attach too high an importance to a case with exaggerated nervous symptoms, where there is an organic lesion of the valves. I have seen many patients lead lives of great restriction, with a certain amount of fear, on account of the supposed seriousness of heart trouble. Women have had attacks of angina pectoris, and have been forbidden to undertake their household duties. Great numbers have gone to health resorts at great expense and inconvenience year after year, to perform the ‘ cure ’, because in their days of suffering they had experienced benefit. When there is a valvular murmur, we must carefully inquire into the conditions that have induced the heart exhaustion and the attendant suffering, and consideration of the whole of the facts will enable us with certainty to recognize the real nature of the phenomena. We can often with certainty reassure the patient that with suitable treatment the suffering will, to a great extent, disappear, and that though the organic trouble may persist, with intelligent management there is good ground for hope of a fair restoration to health. In many cases one is able to do more, to point out that the suffering is a safeguard, its first appearance being an evidence that the patient is exhausting the reserve force of the heart, and that such restrictions are necessary to guard against further exhaustion.

This intelligent appreciation of symptoms is of service in other ways. Thus, when patients become conscious of an extra-systole, they are often subjected to long courses of treatment, usually inefficient, by their medical

attendant, or by some special method. The mere reassurance of the harmlessness of the symptoms would have done more good than all the treatment. As an illustration I cite the following experience. A professional football-player consulted me because his heart 'stopped' at times. He had been seen by two doctors, who forbade him to play, and put him on digitalis and strychnine. This cessation of work was a serious matter to him, because if he could not complete his engagement for the season he lost the chance of a benefit match, which he looked upon as a reward at the end of his services as a footballer. Except that he was frightened and nervous I found him in all respects a healthy man, save for a somewhat frequent extra-systole. I told him he could start playing at once, and that when he was conscious of his heart stopping he was to pay no attention to it. He at once resumed his engagements and completed his term with no discomfort. He told me that at the beginning of one match he was painfully conscious of his heart's irregularity, and felt he must retire, but reflected upon what I had told him, dashed into the game, and in a few minutes forgot all about his trouble, and said he never played better in his life.

I use this illustration to emphasize the fact that neither the exaggerated sensory and mental sensations, nor the symptoms giving rise to them, should be the guide, but what effort the heart can undertake without discomfort.

Besides reassuring the patient, steps should be made to remove him or her from any conditions that conduce to the exhaustion, such as overwork, worry, nursing a sick relative. Discretion must be used, and the patient's circumstances considered. When the patients are well-to-do, a complete change in the mode of life is often very efficacious, and they may be sent away, the choice of the place depending on the patient's tastes. If they can undergo some physical effort, a holiday may be recommended that includes some exertion, such as hill-climbing, cycling, golfing, or 'sight-seeing'—town or country—provided the occupation interests the patient. They may be sent to some watering-place, and may indulge in the special treatment adopted there—for the sake of doing something, and getting what benefit hydrotherapy may convey. My patients have gone to all sorts of places, and those who went to the seaside and indulged in sea-bathing got more benefit than those who frequented the more vaunted inland spas, home or continental. The life there is more bracing, and there is less opportunity to meet all sorts of neurotics, and the baneful habit of comparing experiences is thus not so easy to indulge in.

The vast majority of patients cannot go away and leave their posts, and these form the class with which the general practitioner has often a great deal of trouble. With patience and perseverance, however, much can be

done for them, and in many instances the doctor can give great help to some of the most deserving patients he has to treat. The mother of a family has to keep going, the tired daughter has to nurse the ailing parent. In all these cases there will be found, almost invariably, insufficient sleep, or sleep frequently disturbed, and this is often the real cause of the trouble and in addition renders the patient peevish and irritable. Much can be done by suggesting different devices to promote the patient's rest. In many cases we have to resort to drugs, and happily the most efficacious hypnotic in these cases is also the safest, namely, bromides (particularly bromide of ammonium), which should be given until the patient sleeps soundly. Often they produce drowsiness and languor during the day, and the patient may complain of being weaker than ever. This is no contra-indication, but the contrary, for the languor induces idleness and restfulness. She does less work, she is less irritable, and the heart is not so easily excited. After a few weeks, if the dose be gradually diminished, it will often be found that in the meantime the patient's condition has wonderfully improved. The necessity of continuing her duties prevents the possibility of any immediate recovery, but by the judicious administration of the bromides, patients can be tided over trying periods for months or years.

'Heart tonics' in these cases are of little use. Even if they had the action they are supposed to possess, it is doubtful if their administration would be wise. It is not a whip an overworked horse needs, but rest.

The nourishment of the patient often leaves much to be desired. The household duties and the cooking take away the appetite, and they content themselves with stuff easily swallowed and stimulating—hot fluids, tea, coffee, and spirits. The food should be taken in small quantities and often, and should be fairly dry to ensure slow mastication. It is sometimes a good plan to suggest a dietary of a very simple nature. Find out what food the patient prefers, and, if it is rational, so arrange the diet that every few hours may bring a change, even if it be but an egg at one time, and a dry biscuit and a few tablespoonfuls of milk at another. In all cases stimulants should be forbidden. The great exhaustion brought about by the long weary hours of work and suffering is often speedily temporarily relieved by spirits; but these are just the people who ultimately find solace in increasing quantities, until the habit becomes all too powerful.

CHAPTER IX

Instrumental Methods of Examination

In the examination of the vast majority of patients, the diagnosis can be made independently of graphic records. It must not be inferred from this that graphic records can be dispensed with, for the power to diagnose the great majority of cases comes through the information obtained by this means. Though it is not necessary for a physician himself to take records, he must be familiar with their interpretation in order that he may appreciate and apply the results.

There have been many methods devised to record the movements of the circulation, but here I will only deal with those which in my hands have yielded very satisfactory results. The essence of a method should be simplicity, for the more complicated the processes the more unsuitable it becomes for practical clinical purposes. In hospitals with large staffs of assistants the more elaborate methods may be usefully employed, but for the practitioner who studies his own patients, the simpler the methods the better.

§ 69. **The sphygmograph.**—It is scarcely necessary to enter into a full account of the construction of the various sphygmographs. They have been so frequently described in textbooks that their construction is familiar to all medical men. They are all practically constructed on the same principle. A steel spring is laid upon the radial artery at the wrist in such a manner that, while it compresses the artery, it does not obliterate it. Attached directly to the spring is a long lever, or a series of small levers, that magnify the movements of the spring. The free extremity of the lever presses lightly against a strip of paper, whose surface has been blackened by the smoke of burning camphor or turpentine, the strip of paper passing at a uniform speed by means of a clockwork arrangement. Although I have worked with several instruments, I find the Dudgeon to be the handiest and most useful. Into all sphygmographic records, certain errors, due to

defects of the instruments, creep. Some of the more elaborate instruments may be freer from defect than the Dudgeon, but so long as one is cautious not to read into the tracings movements evidently due to instrumental errors, the Dudgeon sphygmograph is quite serviceable for a great many practical purposes, and more particularly for giving a true and accurate record of the occurrence of pulse beats.

§ 70. **The polygraph.**—There are many perceptible movements due to the circulation that the sphygmograph fails to register, and when it is required to record these movements other instruments have to be employed. The method most commonly adopted has been by conveying, by means of a tube containing air, the movements to be registered to a tambour on which there rested a lever. The excursion of the lever is recorded on a revolving drum covered by smoked paper. Two or more tambours being

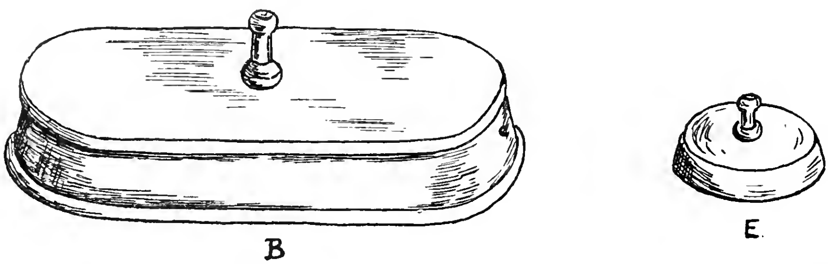

FIG. 13. Shows the shallow cups or 'receivers' used in taking tracings of the liver (receiver *B*) and of the jugular or carotid pulse or apex beat (receiver *E*). (Half size.)

used with their levers placed one above the other, the simultaneous record of different movements can be readily effected.

The elaborate and bulky apparatus required has restricted the employment of this method to such narrow limits, that numerous points of interest in clinical medicine have been either overlooked or misunderstood. In my investigations into the nature of the venous pulse, I had at first to use this unwieldy instrument, but its cumbersomeness compelled me to devise a much simpler and more effective apparatus.

§ 71. **The clinical polygraph.**—This apparatus, which I have called the clinical polygraph, can be used for taking, at the same time and on the same recording surface, tracings of the radial pulse, with tracings of the apex beat, carotid, venous, or liver pulse, or the respiratory movements, and its size permits of it being carried about with the greatest facility, and readily employed in general practice (Fig. 14).

The essential parts of the instrument are a small cup for receiving the impressions of the pulsations, a tube for transmitting the impressions to

a tambour and lever, the tambour being attached to a Dudgeon or Jacquet sphygmograph.

The small cup for receiving the impressions (which will be referred to hereafter as the 'receiver') is simply a small shallow vessel, circular in shape, one and a half inches in diameter and half an inch in depth (*E*, Figs. 13 and 18). The open mouth is applied over the pulsating part so that its edges are closely adapted to the skin and all communication with the outer air is excluded. From the roof of the receiver rises a narrow pipe, half an inch in length. To this pipe is fitted an india-rubber tube three to four feet in length, the other end of which is connected with the tambour. A modification of this receiver is required when tracings of the liver pulse are taken. The 'liver receiver' (*B*, Fig. 13) is larger, being five inches in length, two

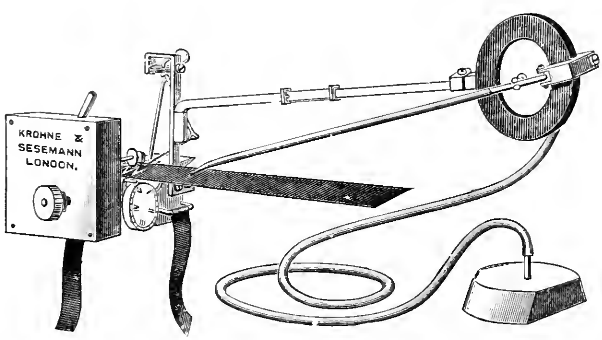

Fig. 14. The clinical polygraph, consisting of a tambour attached to a Dudgeon's sphygmograph.

inches in breadth, and one inch in depth, its open edges slightly curved on their long axis. A small air-hole is made at one end near the roof. In employing the 'liver receiver' the position of the lower margin of the liver having been ascertained, the receiver, held in the right hand, is laid lengthwise across the abdomen, its lower edge being two inches below the liver margin, and the end with the air-hole towards the middle line. Steady continuous pressure is applied to the lower margin of the receiver till it presses deeply into the abdomen, and then the upper margin is adapted closely to the skin. In this manner a considerable portion of the lower liver edge is embraced by the receiver. If the forefinger of the right hand is now applied over the air-hole, the movements of respiration and liver pulse will be communicated to the lever. If the patient stops breathing, the liver movements are alone transmitted.

The tambour (Fig. 15) supports a writing lever about six inches in length. From the under surface of the tambour a pipe protrudes, which is connected

by the india-rubber tubing with the receiver. Screwed tightly to the bottom of the tambour is a stem (*B*) six and a half inches in length, projecting outward parallel to the under surface of the tambour. Half an inch of the other extremity of the stem is bent almost at right angles (*C*), and this portion fits into a slot on the upright stem that supports the movable lever of a Dudgeon's sphygmograph (Fig. 14). When the tambour arrangement is adjusted to the sphygmograph, the tambour rests with its movable surface vertical, so that the writing lever moves horizontally. For the purpose of adjusting the point of the writing lever to any desired place on the recording paper the following movements are available : The point of the lever can be approximated to, or withdrawn from, any desired point by means of a sliding arrangement at *D* (Fig. 15), where the stem consists of two parts clasping one another. The writing point can be made to move in the horizontal direction, so as to write at any desired level on the paper, by means

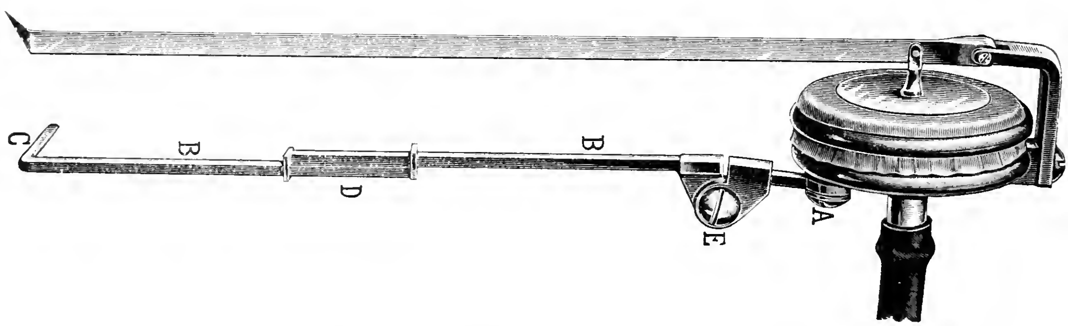

FIG. 15. The tambour with stem for attachment to the Dudgeon or Jacquet sphygmograph.

of a joint at *E* (Fig. 15), which moves stiffly, and retains the position in which it is placed. Finally, by rotating the tambour at a joint formed where the stem is fixed to the under surface of the tambour, the lever can be raised out of reach during the adjustment of the sphygmograph to the pulse, and brought back and allowed to touch the surface of the blackened paper, with sufficient delicacy to permit its movements to be accurately recorded without being restrained by too close pressure. By means of these arrangements the lever can be moved with great facility and accuracy in a vertical and a horizontal direction, as well as made to approach or recede from any given point. By this method any movement can be recorded at the same time, and on the same paper, as the radial sphygmograph. One can make the tambour lever write directly above or below the writing lever of the sphygmograph, or, when the movements are large, a little behind, so that the two levers do not come into contact during their excursions. Perpendicular lines to show the relative time of the two tracings are obtained by allowing

the lever to make distinct marks on the paper before starting or after stopping. With a pair of compasses, the relative time of any event can be accurately gauged.

Some difficulty may be experienced in the employment of the clinical polygraph, on account of the weight of the tambour tilting the sphygmograph off the radial pulse, especially if the inelastic band usually supplied with the sphygmograph be employed. I have long ago abandoned the use of the inelastic band (placing no reliance on the pressure supposed to be required to obtain an idea of the arterial pressure), and employ instead an elastic band, tying the instrument to the wrist by a knot. Should any slipping occur, the sphygmograph and tambour can readily be adjusted. It frequently happens that the radial pulse causes such a large excursion that there is no clear space on the paper for the venous or other pulsation.

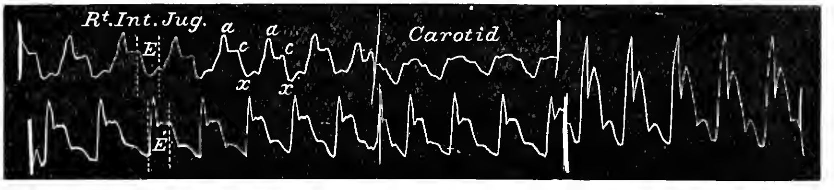

FIG. 16. The first part of the tracing shows the venous pulse from the right internal jugular vein (upper tracing) taken at the same time as the radial pulse (lower tracing). The clockwork being stopped, the receiver was applied over the carotid artery, and tracings of the carotid and radial pulses taken. Again the clockwork was stopped, and the tambour portion of the polygraph removed, and the full effect of the radial pulse was inscribed on the last portion.

In such a case I either shift the sphygmograph till I obtain a tracing of less amplitude (seeing that the object of taking the radial pulse at the same time as the venous is to obtain an idea of the relative time), and, before the paper has entirely passed through, stop the clockwork, remove the tambour, and obtain a full-sized tracing of the radial pulse (Fig. 16). Or the venous pulse may be taken a little behind the radial, and the various events disentangled. For accurately estimating the time of the events, it is advisable to take on the same paper a few beats of the carotid pulse. For convenience of study, other events can be readily recorded within the limits of a short sphygmographic paper; and a record of apex beat, venous, carotid, and liver pulse may be taken at the same time as the radial sphygmogram (Fig. 17).

When a long tracing is required (as, for instance, in noting the movements of the two sides of the heart in cases of occasional irregularity), a tracing paper one or two feet in length may be employed.

For convenience, I have had the Dudgeon sphygmograph altered so as to take a paper over one inch in width, thus allowing more room for the two tracings. At one time I had attached a time-marker, driven by a separate clockwork as in the Jacquet, but this added too much to the weight of the machine, so that it was easily displaced. Biggs has recently devised a convenient little time-marker which can be fixed to a Dudgeon or other sphygmograph.

I have enlarged the box for holding the blackened paper, by adding a compartment to contain the tambour, so that it is conveniently carried on a visiting round.

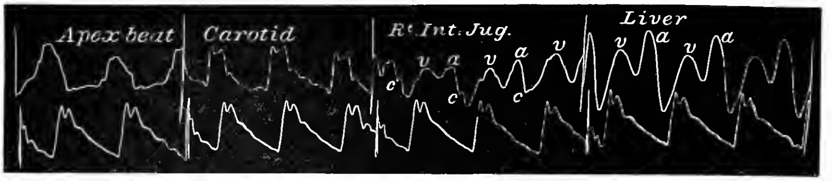

Fig. 17. Apex beat, carotid, right internal jugular, and liver pulses (upper tracing) taken at the same time as the radial pulse (lower tracing).

§ 72. **The ink polygraph** (Fig. 18).—Simple and useful as is the instrument just described, I found it was not convenient when the movements had to be recorded over a long period, as where the irregularities were infrequent, or where they varied, or where respiratory curves were required. I therefore conceived the idea of constructing an instrument that would take tracings of an indefinite length, where the employment of ink would enable a roll of paper to be unwound, and save as well the inconvenience of blackening and varnishing.

I had considerable difficulties to overcome, but found a skilled helper in Mr. Shaw, who not only comprehended and appreciated my ideas, but constructed an instrument that carried them out. The case *A* (Fig. 18) contains the clockwork for the roller which unwinds the roll of paper *D*, and also the separate clockwork which moves the time-marking pen *F*. *B B* are the two tambours, and *F F* their levers. The writing pens in Fig. 18 are narrow-grooved wires, one end fixed to the bottom of a small cistern at the free extremity of the lever. The other end of the grooved wire is adjusted to barely touch the paper. The ink is put into the tiny cistern, and it flows along the groove to the pen point by capillary attraction. Recently these pens have been greatly improved by making a shallow groove in the lever act in place of the cistern. If the pens are kept clean, and the ink is free from dust, they serve their purpose most admirably, and are ever ready for use. Red ink is better than the black inks, as it

does not corrode the pens. As the radial pulse is the most serviceable of standards, a special method is employed to record it. A splint (*C* 1) is fastened to the wrist in such a manner that the pad of the steel spring falls on the radial artery, and is pressed down by an eccentric wheel (18) until a suitable movement is transmitted to the spring by the artery; then the broad tambour (*C*) is fitted on to the splint so that the knob (12) falls on the moving spring. This wrist tambour is connected to the tambour *B* by india-rubber tubing (22, 22), and the movements of the

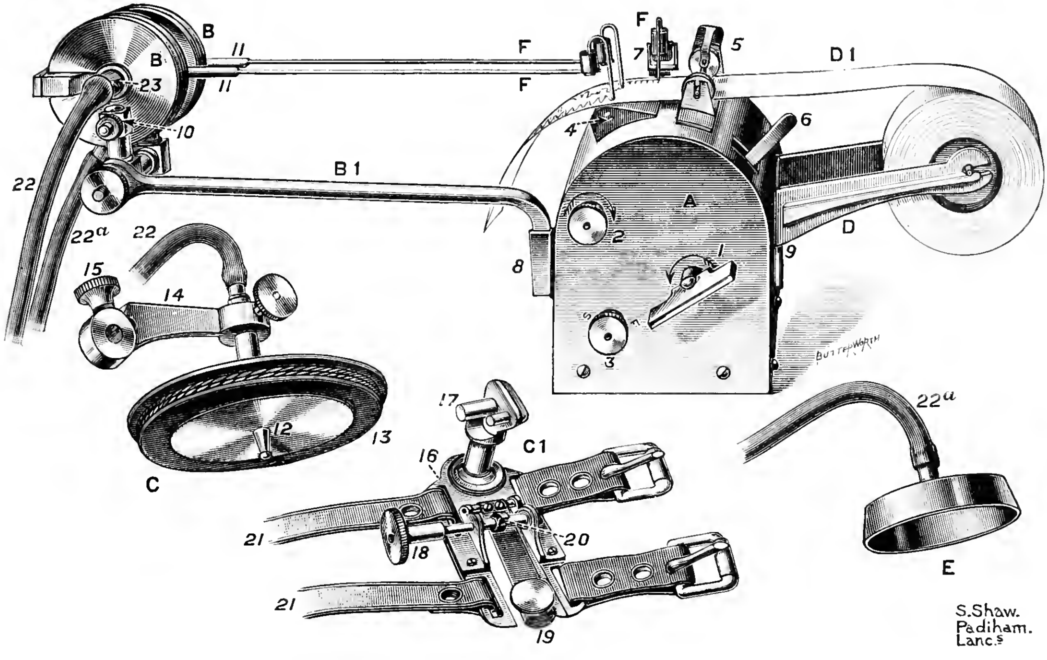

FIG. 18. The ink polygraph.

radial pulse are recorded by the lever *F*. The shallow cup (receiver) *E* is placed on the pulsation which it is desired to record, and the movement is conveyed to the lever *F* of the other tambour. In this way, simultaneous with the radial pulse, a record can be obtained of the apex beat, carotid, jugular, or other pulses.

To record the respiratory movements, a bag can be substituted for the receiver *E*.

By turning the screw (3) the rate at which the paper passes can be quickened or slowed at will. This is of the greatest use, for it often happens that in quickly succeeding events a wider interval may be required, whereas in recording respiratory movements a slow rate is best. As the time-marker

registers one-fifth of a second, and is driven by a separate clockwork, the rate of the recorded movements can always be ascertained with absolute accuracy.

It has been suggested that another tambour should be added to record a third movement, and I have tried this but have practically discarded it, as, though it might be of use occasionally, it would complicate the apparatus unnecessarily. When one is making observations single-handed, the two tambours are quite sufficient to occupy the attention. With a little practice this apparatus can be used with the greatest facility. In the course of a few minutes the different movements can be recorded with the patient sitting up or in the recumbent position.

When the tambour is strapped to the wrist to take the radial pulse, one hand is always free to start the machine, and to replenish the ink or regulate the rate, the other hand holding the receiver over the movement to be recorded.

The tracings in all the figures in the Plates except Figs. 90 and 103, and many in the text, have been taken by this instrument.

CHAPTER X

The Position and Movements of the Heart

§ 73. **The position of the heart in the chest.**—The position of the heart in the chest, and the relations of its chambers to the chest-wall, can best be realized from such a drawing as Fig. 19. The chief points to notice are the position of the apex and how it is made up of the left ventricle and covered normally by the lung. The whole right border of the heart is made up by the right auricle, and the greater portion of the anterior surface by the right ventricle. The perceptible movements of the normal heart when covered by the lung are due to the right ventricle, and this is the reason that the apex beat, due to the left ventricle, is not obtained from people with voluminous lungs.

§ 74. **The standards for recognizing the events in a cardiac revolution.**—Owing to their easy recognition and determined place in the cardiac cycle, the carotid and radial pulses form the most certain standards for finding out the place of other movements in a cardiac revolution. In describing the tracings frequent references will have to be made to these standards, and more particularly to that period during which the semilunar valves are open, which is indicated in the tracings by the space *E*. When it appears in the radial tracing it corresponds to the effects of the ventricular systole upon the radial pulse—that is to say, to the actual pulse-wave—and

not to the true time of the occurrence of the ventricular systole, for the pulse-wave having a longer distance to travel, the period *E* will be later in the radial than in the apex or carotid tracings.

§ **75. Conditions of the chest-wall permitting the recognition of certain movements of the heart.**—The movements of the heart in a healthy person are often so obscured by the lungs, that only very little change is

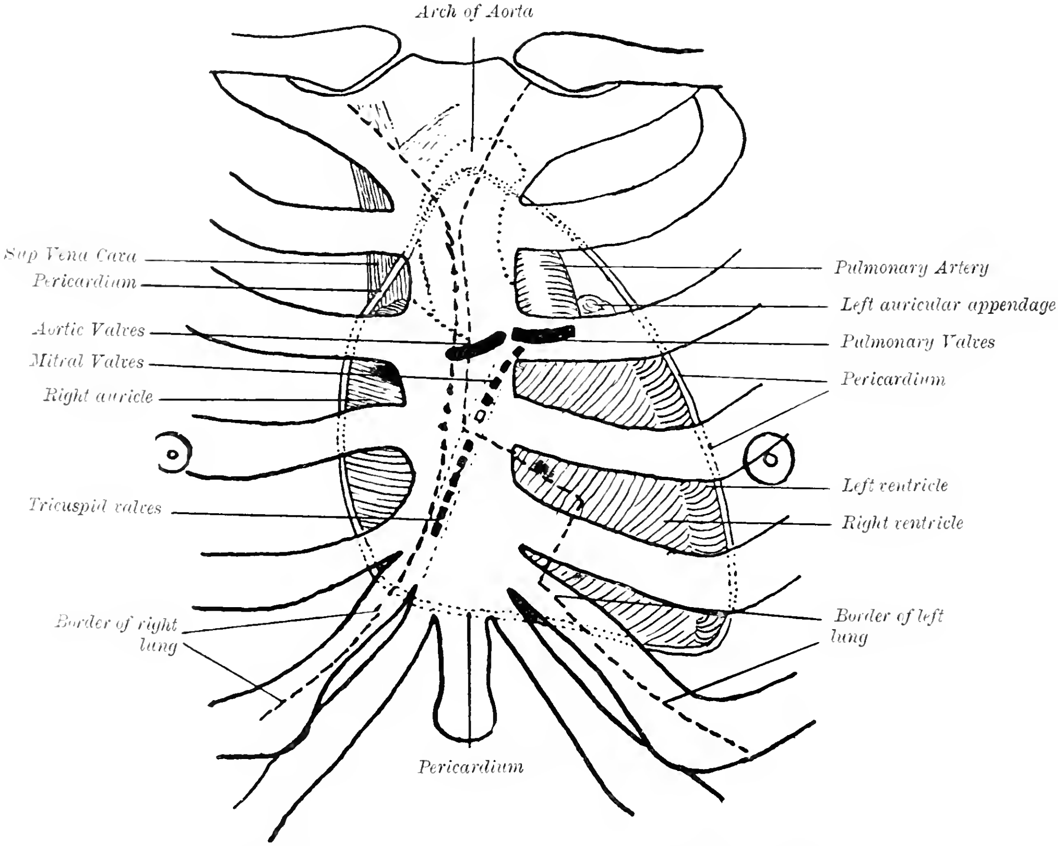

FIG. 19. The position of the heart in the chest. (Keith.)

discernible in the external chest-wall. In many cases the lungs are so voluminous, or the chest-wall so fat and thick, that no movement can be detected. But when a large surface of the heart is directly exposed to a thin chest-wall, the heart being normal or increased in size, and the lung displaced, a series of movements of the chest-wall, due to the contraction and expansion of the heart, can be recognized. The movements thus discernible are not the same in all cases, but depend on which part of the heart's surface comes in contact with the chest-wall, and other yielding structures. These move-

ments take place so rapidly that it is difficult to interpret their significance by the unaided senses. Many writers on this subject have drawn elaborate, but nevertheless erroneous, conclusions from such unaided observations, and it seems to me that accurate observations by the graphic method alone can furnish a clear and definite explanation.

§ 76. **The nature of the movements graphically recorded.**—The movements of the heart that are most readily recognized are those connected with the systole and diastole of the ventricles. Movements directly due to the auricles are so obscured by the larger and more vigorous movements of the ventricle, that it is doubtful if they are ever capable of recognition. The movements most readily recognized are: first, the apex beat; second, the filling of the ventricles; third, the emptying of the ventricles; and fourth, the shock communicated by the sudden hardening of the ventricular walls as they pass into systole.

§ 77. **The apex beat.**—The chief movement, and the one which is usually most apparent, is that caused by the forcible outward projection of the apex of the heart during the ventricular systole—the apex beat. This is generally described as 'the lowest and outermost point of the heart which strikes against the chest-wall'. In healthy adults, it is usually felt in the fifth left intercostal space, immediately inside the nipple line. It may, however, be situated in the fourth interspace, and outside the nipple line in children, and in some adults. In disease of the heart it alters its situation with the increasing size of the heart. This forward thrust occurs when the left ventricle is in contact with the chest. As will be shown later, a movement of another description occurs when the right ventricle constitutes the so-called apex beat. That the apex beat due to the left ventricle is a distinct displacement of the heart forward can be recognized by the senses of touch and sight.

During the whole time occupied by the systole of the ventricle, the apex is usually kept projecting into the interspace, so that the palpating hand recognizes the forward thrust, and in such a tracing as Fig. 20, the lever taking the tracing is kept raised during the whole time of the outflow from the ventricle (space E'). If the left ventricle is much hypertrophied, the same movement can sometimes be detected in two or three interspaces. If the interspaces be fairly open and the chest-wall thin, and the tip of the finger be thrust into the third or fourth interspace near the sternum, the right ventricle can be felt hardening, and remaining thus hardened in contact with the finger during the whole period of the ventricular systole. It cannot, however, be averred that in this case there is a forward thrust. The heart here is always in contact with the chest-wall, and the finger pushed into the

interspace during the diastole in all likelihood impinges against the lax ventricular wall. As soon as the ventricle hardens, the finger recognizes this hardening as something pushing against it. This sensation of a thrust is sometimes actually synchronous with an indrawing of the soft structures filling up the interspace (Fig. 26).

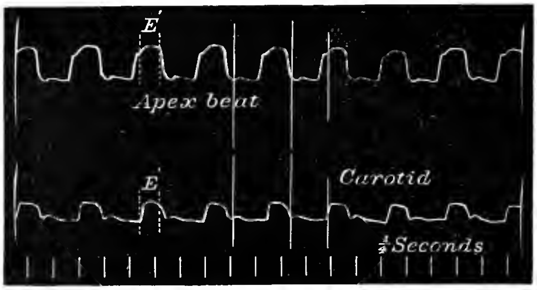

FIG. 20. Simultaneous tracings of the apex beat and the carotid pulse, showing the 'systolic plateau' in the cardiogram during the outflow from the ventricle (E').

§ **78. Interpretation of a tracing of an apex beat due to the systole of the left ventricle.**—A tracing of the apex beat or cardiogram is a diagrammatic representation of (a) the forward movement of the apex of the heart while the ventricular muscle is beginning to contract (space D, Fig. 21);

FIG. 21. Simultaneous tracings of the apex beat and of the pulsation in the pulmonary artery. a represents the small wave due to the auricular systole. The time during which the ventricle is passing into systole is represented by the space (D), emptying (E), relaxing (F), filling (G). While this tracing was being taken the cylinder was rapidly rotated. The letters D, E, F have reference to the same periods in the cardiac revolution as in Fig. 43 (see p. 108).

(b) the retention of the apex beat against the chest-wall while the ventricles are emptying (space E, Fig. 21); (c) the backward movement of the apex of the heart while the ventricular muscle is relaxing (space F, Fig. 21); (d) and the gradual swelling of the ventricle during diastole (space G, Fig. 21).

(a) *The period of commencing contraction of the ventricular muscle—the presphygmic interval* (space D, Fig. 21).—During this period the pressure

within the ventricle is rapidly rising. The auriculo-ventricular valves close as soon as the pressure within the ventricle rises above that in the auricle, and the semilunar valves open as soon as the pressure in the ventricle rises above that in the aorta. This last occurs at the end of the period *D*, Fig. 21, and is usually indicated by the abrupt termination of the upstroke.

In Fig. 21 simultaneous tracings were taken of the pulsation in the pulmonary artery and of the apex beat. As the beginning of the pulse in the

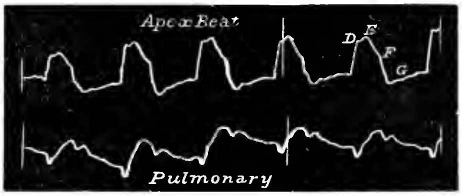

FIG. 22. Simultaneous tracings of the apex beat and of the pulsation in the pulmonary artery. The letters have the same significance as those in Fig. 21.

pulmonary artery indicates the opening of the semilunar valves, so it is found that the end of the period *D* corresponds exactly with the beginning of the pulsation of the pulmonary artery. When this figure was taken the cylinder was rotated rapidly in order to separate the events as widely as possible. When the cylinder rotates at a slower rate, this period is represented by an almost perpendicular line (Fig. 22).

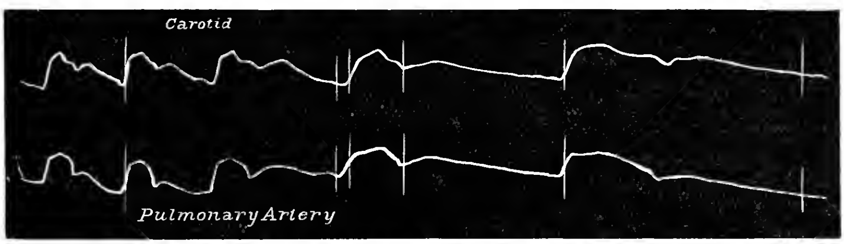

Fig. 23. Simultaneous tracings of the pulses of the carotid and pulmonary arteries. After the third beat the cylinder was rotated rapidly.

It will be noted that the termination of the upstroke corresponds exactly with the beginning of the outflow of the ventricle into the artery. These tracings were taken from a lad suffering from phthisis of the left lung, which had retracted from the heart, and through the thin chest-walls the various movements could easily be observed. In the second left interspace there was a marked pulsation, and tracings of this, taken at the same time as the carotid pulse, left no doubt as to its being caused by the pulmonary artery (Fig. 23). It will be noticed that the carotid pulse appears just a very little

later than the pulmonary. Here also, after a few beats, the cylinder was rapidly rotated with the hand to separate more widely the different events.

(*b*) *The period of ventricular outflow* (space *E*, Fig. 21).—When the pressure in the ventricles exceeds that in the aorta and pulmonary artery the semilunar valves open, and the blood flows out from the ventricles. During this period the apex is usually kept stationary, pressing against the chest-wall, and in many tracings (as in Fig. 24) it is shown by a fairly level line—the systolic 'plateau'. In place of a flattened top representing the period of ventricular outflow, the tracing may continue to rise (as in Fig. 20), indicating that the ventricle is still slightly shifting. On the other hand, the tracing sometimes rapidly descends (Figs. 21 and 22). I cannot but think that this is due to the ventricle shrinking away from the interspace during its systole, the receiver perhaps not being exactly over the apex. I shall show later on that this shrinking can be demonstrated in various places, and I have found evidence of it immediately under a diffuse apex beat. The movement producing the apex beat is really a displacement of the heart forward, and while the heart is thus displaced the ventricles shrink as they empty themselves (see § 20). The termination of the ventricular outflow is occasioned by the pressure in the aorta becoming higher than that in the ventricle. The semilunar valves close in consequence, and the ventricular muscle then relaxes; the termination of the systolic period is indicated in the cardiogram by a sudden descent.

(*c*) *The period of relaxation of the ventricular muscle* (space *F*, Fig. 21). —With the relaxation of the ventricular muscle the apex retreats from the chest-wall, as is indicated by the slanting downstroke in the tracing, or where the tracing is already falling during the ventricular outflow by a more rapid descent (Figs. 21 and 22). During this period the ventricular pressure rapidly falls until the stage of complete relaxation, when the pressure inside the ventricles becomes lower than that inside the auricles. When this occurs the auriculo-ventricular valves open. The apex then has reached its greatest distance from the chest-wall, and in the tracing the lowest point is reached.

The time of the opening of the auriculo-ventricular valves is usually a very definite landmark in apex and jugular tracings, and, in consequence, is a useful standard for measuring the sequence of events in tracings of irregular heart action. It is recognized as the lowest point reached in tracings of the apex of the left ventricle, and it is just before the fall of the wave *v*, in tracings of the jugular pulse. Its time corresponds nearly with the bottom of the aortic notch in tracings of the radial pulse. It is represented by the perpendicular line 6 in many of the tracings given later.

(*d*) *The period of filling of the ventricles* (space *G*, Fig. 21).—Upon the opening of the auriculo-ventricular valves, the blood flows from the auricles into the ventricles, and as the ventricles distend the heart pushes against the intercostal space, and slightly raises the lever. This period is marked in the tracing by a gradual ascent. Frequently, however, the heart fails to affect the tissues in the interspace during this period, so that no indication of the filling of the ventricles is obtained ; in such a tracing as Fig. 20, for example, and in many others given in the text, the whole of this period is a blank, so far as information regarding events in the cardiac cycle are concerned.

§ 79. **The auricular wave.**—In some tracings from the apex there is occasionally found an abrupt though slight rise immediately preceding the beginning of the ventricular systole (*a*, Figs. 21 and 24). This is due to

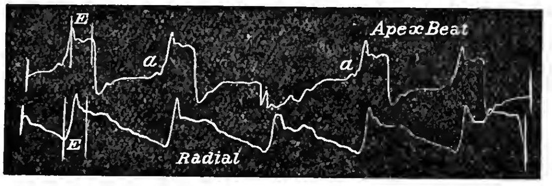

FIG. 24. Simultaneous tracings of the apex beat and of the radial pulse, showing the 'systolic plateau' and the small wave (*a*) due to the auricular systole. The third beat in the apex tracing is obliterated by the movement of inspiration.

a sudden increase in the contents of the ventricle caused by the contraction of the auricle, and may be termed the auricular wave.

The auricular wave is not always perceptible in apex tracings, but when present it often gives valuable information. Normally, it precedes the beginning of the wave due to the ventricular systole by about one-tenth of a second (space between 1 and 2, Fig. 25). Sometimes this interval is increased, and then it may indicate a delay in the passage of the stimulus from auricle to ventricle (Fig. 231, p. 339). In cases of heart-block, it may be recognized during the ventricular pauses (Fig. 123). Its absence may be of no significance, but it is to be noted that it is never seen in cases with the nodal rhythm, even when immediately before the starting of this abnormal rhythm it had been a conspicuous feature (compare Fig. 198 with Fig. 202, p. 332).

§ 80. **Retraction of yielding structures in the neighbourhood of the heart during ventricular systole.**—When the ventricles expel their contents they must of necessity shrink. This shrinkage occurs abruptly, and with considerable force. The yielding tissues in the neighbourhood of the heart are dragged upon, and evidence of this dragging can be obtained

from a variety of sources. John Hunter [338] originated the idea that the systole of the ventricles would have a tendency to produce a vacuum, and thus expedite the flow of the venous blood into the chest. Evidence of this 'cardiac aspiration' affecting the lungs has been obtained by a number of observers. The tracings [167] of Mosso and Delépine [391] of the movements of

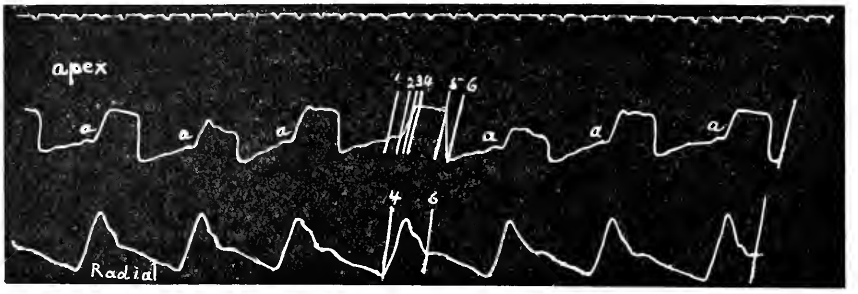

Fig. 25. The apex tracing shows a slight wave, *a*, due to the contraction of the auricles distending the ventricles and beginning one-tenth of a second before the ventricular systole. For explanation of the numbered perpendicular lines see Fig. 46.

the column of air in the respiratory passages, due to the cardiac aspiration, correspond exactly with those obtained from the praecordium (Fig. 26), from under the liver (Figs. 27 and 28), and from the epigastrium (Figs. 29 and 30).

Fig. 26. Simultaneous tracings of the heart movements (upper tracing) and of the radial pulse. The first part of the upper tracing was taken from the apex beat in the fourth interspace immediately outside the nipple, while the latter part was taken in the same interspace near the left border of the sternum. In the first part the cardiogram shows a 'systolic plateau' during the ventricular outflow (*E*), in the other part the cardiogram is inverted, i.e. there is a depression during this period (*E*).

In Fig. 26 this drawing-in of tissues in the intercostal spaces over the heart is demonstrated. It was obtained from a boy aged fourteen. The apex beat was well marked in the fourth interspace outside the nipple. At the same time that the apex was thrust outwards, the skin and subcutaneous tissues over the same interspace inside the nipple were drawn in. In Fig. 26 the tracings of the apex beat were taken simultaneously with the radial

pulse for four beats. The clockwork was then stopped, and the receiver, which had been applied over the apex, was placed over the praecordium inside the nipple, and the 'inverted cardiogram' of the last portion was obtained. The space *E* represents the duration of the outflow from the ventricle; and this period, which in the apex tracing shows a flattened elevation, shows a great depression in that obtained from the front of the heart. The ascending limb of the apex tracing corresponds to the period during which the ventricle is contracting (space *D*, Fig. 21). This period in the inverted cardiogram is represented by a slight rise, due to the shock of the contracting ventricle. No blood as yet has escaped from the ventricle. As soon as the semilunar valves open the blood rushes out of the ventricle, the ventricles diminish in size and the yielding tissues of the interspace sink in and cause the great fall, as represented in the inverted cardiogram (space *E*, in the latter half of Fig. 26).

§ **81. Liver movements due to cardiac aspiration.**—Not only can this aspiration be demonstrated as affecting the pliable tissues immediately in contact with the heart, but in suitable cases it can be shown to produce a distinct excursion of the liver. All writers referring to this movement of the liver speak of it as a downward thrust during the ventricular systole. Careful tracings demonstrate that this movement is quite of the opposite nature—it is a drawing-up of the liver during the ventricular systole. In Figs. 27 the apex beat is taken at the same time as the movement of the liver.

The receiver taking the liver movement being applied to the under surface of the organ, a retraction of the liver upwards corresponds with a fall in the tracing, and vice versa. It will be seen that the movement of the liver upwards takes place during the ventricular systole, while the downward movement is due to the diastolic filling of the ventricle. In Fig. 28 the movement of the liver is recorded at the same time as the carotid pulse. It is seen that as soon as the carotid pulse appears, the liver is drawn up, and remains there until the end of the ventricular systole, after which the liver gradually falls down. I do not mean that the excursion of the liver is one of considerable extent, but the movement is so great as to be obvious to the palpating hand. It is distinct from a pulsation of the liver, which is a periodic swelling of the liver, while this is a displacement of the liver *en masse*.

§ **82. Epigastric pulsation.**—The causes which may produce a pulsation in the epigastrium are: (*a*) a dilated right heart; (*b*) a hypertrophied left ventricle; (*c*) the abdominal aorta; and (*d*) an aneurysm of the abdominal aorta.

In the later stages of typhoid fever and other exhausting diseases, epigastric pulsation is an ominous sign of cardiac enfeeblement. The movement consists of an alternate swelling and retraction of the epigastrium. It is invariably assumed that this swelling or pulsation is due to the right

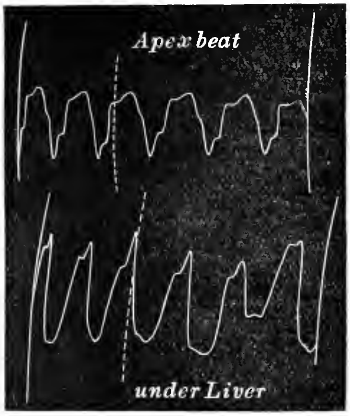

FIG. 27. Simultaneous tracings of the apex beat and of the movement of the liver. When the ventricle empties the liver is drawn up, and this causes the fall in the tracing.

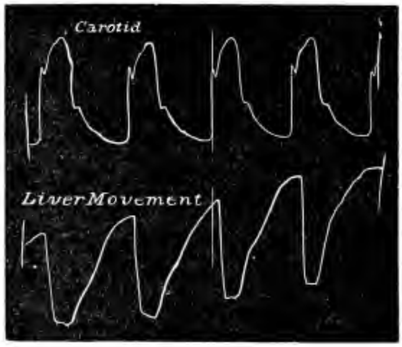

FIG. 28. Simultaneous tracings of the carotid pulse and liver movement. With the appearance of the carotid pulse there is a sudden fall of the lower tracing due to the liver being drawn upwards by the emptying ventricles.

ventricular systole, and that it is of the same nature as the outward protrusion constituting the apex beat. If this form of epigastric pulsation is carefully timed with the carotid pulse, it will be found that the epigastric pulse, protrusion, or swelling, precedes the carotid pulse, and that the retraction of

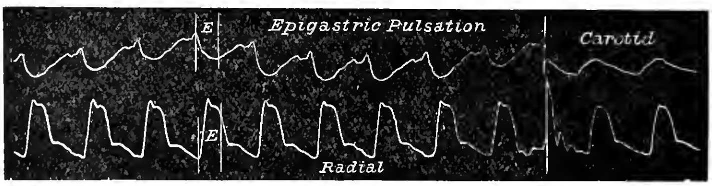

FIG. 29. Simultaneous tracing of the epigastric pulse, due to a dilated right heart, and of the radial pulse. The epigastric pulse shows a retraction during the ventricular systole (*E*), and a protrusion during the filling of the ventricle.

the epigastrium corresponds in time to the carotid pulsation. The apex beat is rarely available in these cases, on account of the right heart pushing the left ventricle backwards. In the tracings of the epigastric pulse (Fig. 29) the radial pulse is taken as the standard of time. The time occupied

by the pulse travelling from the heart to the wrist being allowed for, it will be found that the great fall in the epigastric pulse corresponds exactly with the ventricular systole (E).

The patient from whom this tracing was taken was dying from pernicious anaemia. At the post-mortem examination, a needle pushed through the epigastrium, at the place where the tracing was obtained, was found to have penetrated the right ventricle.

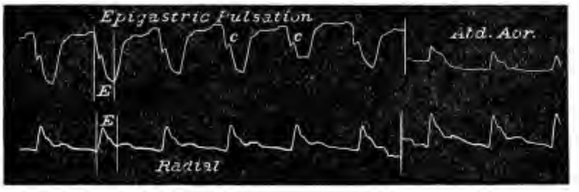

FIG. 30. Shows the same features as Fig. 29, with the exception of the small wave (c) occasioned by the shock communicated to the epigastrium by the abdominal aortic pulse. A few beats of the abdominal aorta are also given.

In Fig. 30 a similar tracing is given, except that there is a slight interruption at c, on the line of descent. This will be found to correspond exactly to the time of the abdominal aorta, taken from the middle of the abdomen, a few beats of which are also given. This small wave (c) is due to the impulse imparted to the tissues by the pulse of the underlying aorta. Epigastric pulsation, due to hypertrophied left ventricle, has the same character as an apex beat (Fig. 31). Epigastric pulsation due to the abdominal aorta

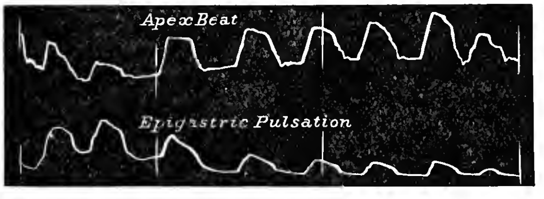

FIG. 31. Simultaneous tracings of the apex beat and of the epigastric pulsation, due to a hypertrophied left ventricle.

presents quite a different character from that due to a dilated right ventricle, as shown in Fig. 32, where the pulse corresponds in character and time with the radial pulse. An epigastric pulsation due to an aneurysm of the abdominal aorta would assume the time and character of the abdominal aortic pulse (Fig. 32).

§ 83. **The apex beat due to the right ventricle.**—Accepting the usual clinical definition of the apex beat ' being the lowest and outermost part of the heart's impulse ', a totally different form of beat is found when the right

ventricle causes this movement. In certain cases of dilatation of the right heart, nearly the whole anterior aspect of the heart is composed of the right auricle and ventricle, the left ventricle forming but a mere strip of the border (see Figs. 135 and 136). This portion of the left ventricle is situated so far back that it is covered by the lungs, and does not reach the chest-wall. Hence it is that 'the lowest and outermost part of the heart' in contact

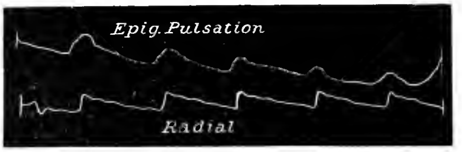

FIG. 32. Simultaneous tracings of the epigastric pulse, due to the abdominal aorta, and of the radial pulse.

with the chest-wall is the right ventricle. The character of the apex beat now corresponds exactly with that of the liver movements, of the epigastric pulse, due to enlarged right heart, and of the inverted cardiogram in Fig. 26. In place of the outward thrust during the systole, as in the apex beat due to the left ventricle, there is an indrawing of the tissues.

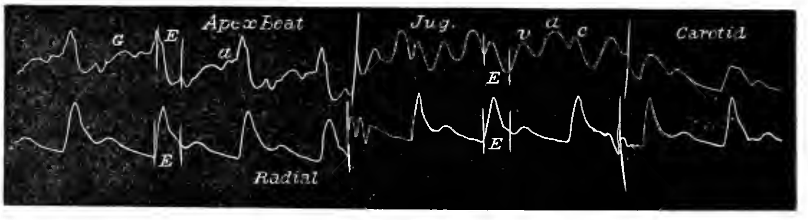

FIG. 33. Tracings of the apex beat, jugular pulse, and carotid pulse (upper tracing), taken at the same time as the radial. The apex tracing is due to the right ventricle, and shows a depression (E) during the ventricular outflow. The sharp elevation preceding E is caused by the shock of the contracting ventricles. This is preceded by a small wave (a), which is due to the contracting auricle distending the ventricle, and corresponds in time exactly with the wave (a) in the jugular pulse, which is due to the contracting auricle sending a wave of blood back into the veins.

Fig. 33 was taken from a youth, aged eighteen, with simple dilatation of the heart, and free from valvular disease. There was marked pulsation of the jugular veins, a few beats of which are given. The apex tracing shows a great depression during the period of ventricular outflow (E). This period is immediately preceded by an abrupt rise due to the shock communicated to the chest by the sudden hardening of the ventricular wall. Although corresponding with the period D (Figs. 21 and 22) in the left ventricular apex tracings, I am inclined to think that the rise here is an instrumental

fault, due to the violent shock communicated by the sudden and forcible ventricular contraction. This period is preceded by a small wave (a) in the tracing identical with the similar rise in Figs. 24 and 25 of the left ventricular apex beat. It is due in this case, as in those, to the distension of the ventricle by the auricular systole. It occupies exactly the same period in the cardiac revolution as the wave (a) in the venous pulse, which is produced by the systole of the right auricle. The space E in all the tracings represents the period of ventricular outflow as it affects the different pulses. One can, therefore, readily and with certainty refer the different events to their causes. Thus we know that the wave of contraction arising in the auricle passes on to the ventricle, that between the auricular outflow and the ventricular outflow a period, the presphygmic (D, Fig. 21), exists, during which the ventricle is contracting and raising its pressure until it opens the semilunar valves. Thus the presphygmic period in the apex tracing exactly corresponds to the period between the summit of the wave a in the venous pulse due to the auricular systole, and that of the wave c due to the carotid pulse. A few beats of the carotid are given, which can be taken as a standard of time to verify all these points. The period G is due to the filling of the ventricle.

§ 84. **Significance of the inverted cardiogram.**—It is asserted in textbooks that an indrawing of the apex during systole of the ventricles is a diagnostic sign of adherent pericardium. I have had several cases where I have got at one time tracings of the apex beat due to the left ventricle, and at other times tracings due to the right ventricle, with indrawing during systole; and at the post-mortem examination there has never been found any signs of adherent pericardium.

The fact that 'the lowest and outermost point of the heart which strikes against the chest-wall' may be due to the right ventricle should be borne in mind. Whenever that occurs, the cardiogram is an inverted one—that is to say, there is a shrinking of the heart from the chest-wall during the systole and a protrusion during the diastole of the ventricles. This is not always recognizable at first sight. Being somewhat familiar with the form of various apex-beat curves, I generally have no difficulty in recognizing cardiograms due to the left ventricle. But when from a patient I took Fig. 34, I certainly was misled in the first instance. The abrupt rise and fall bear a close resemblance to an apex-beat curve due to the left ventricle. Careful measurements of the radial and apex tracings show that the elevation in Fig. 34 was not at the period of ventricular systole, but occurred during ventricular diastole, while the systolic period (E) corresponded with the fall in the tracing. It is necessary to insist upon this view, because inferences drawn from the apex beat alone are liable to lead one into error.

Even so careful an observer as Keyt[157] has mistaken the nature of such an apex-beat tracing, and imagined in consequence that he detected an extreme delay in the appearance of the arterial pulse. I have observed similar errors of interpretation in the tracings of other writers. It follows then that for a guide to any event occurring during a cardiac revolution the arterial pulse is the only safe and reliable one. When the apex beat is taken as a standard, careful inquiries should be directed to ascertain its true nature. While it is true in the majority of instances that the cardiogram from the right ventricle is 'inverted', I have taken tracings with a systolic plateau from the third and fourth interspaces near the sternum, but as I have had no post-mortem examination in these cases I am not sure of the part of the heart producing these curves. The whole subject of cardiography is in great need of thorough and painstaking investigation.

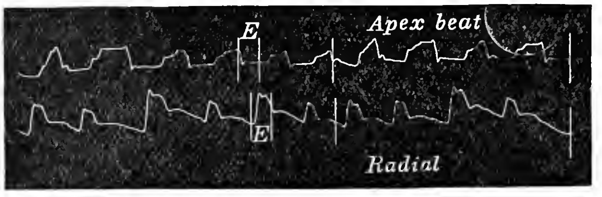

FIG. 34. Simultaneous tracings of the apex beat and of the radial pulse. The rise in the apex tracing resembles the usual characters present in a tracing of the apex beat due to the left ventricle. On analysis it is found that the elevation is during the diastole, and the fall (E) during the systole, of the ventricle.

§ 85. **Alteration of the apex beat due to retraction of the lung.**—If one watches the progress of a case of advancing heart failure over a period of years, marked changes will sometimes be detected not only in the character but in the position of the apex beat. In the earlier stages of heart failure, due to mitral disease, for instance, the left ventricle may be pushed back by the distended right ventricle, so that it is entirely covered by the lung, and the apex beat may then be due to the right ventricle. In course of time, from pressure of the enlarged heart, the lung is compressed and recedes, leaving a large surface of the heart bare to the chest-wall. In such cases the apex beat may be found in the posterior axillary line and in the eighth interspace. The tracing obtained then is one due to the left ventricle.

§ 86. **The shock due to the ventricular systole.**—I am of opinion that a good deal of confusion in regard to the correct interpretation of the heart movements has arisen from associating the shock conveyed to the chest-wall, when the ventricles pass into systole, with the apex beat. The apex beat and this impulse have become so connected that it is assumed

that they are one and the same thing. The apex beat due to the left ventricle is a movement which lasts during the whole of the ventricular systole; the shock caused by the ventricular contraction endures but a short space of time, and occurs while the ventricular muscle suddenly hardens and corresponds with the upstroke only of the apex beat (*D*, Figs. 21 and 22). It is this shock which sends the lever so high in Figs. 24 and 33, at the beginning of the ventricular contraction. In the tracings of the epigastric pulse (Figs. 29 and 30), and of the movement from the front of the heart (Fig. 26), this shock causes the sharp elevation just before the fall (*E*) due to the emptying of the ventricle. Thus, in noting the time of the shock and watching the epigastric pulse, for instance, as in Figs. 29 and 30, one could see that the retraction of the epigastrium followed it. If one associated the shock with the apex beat, it would therefore be assumed that the protrusion corresponded with the systole, and the retraction with the diastole. It frequently happens that this shock is the only movement of the heart discernible on examining the chest. It is often markedly present in dilatation of the heart, when the heart's surface in contact with the chest-wall is entirely made up of the right ventricle and auricle. In such cases it must not be assumed that the shock is the evidence of the contraction of the right ventricle only. It is impossible to distinguish the shock due to the right ventricle from the shock due to the left. The reason I insist upon this is because the perception of this shock has been assumed to be an evidence of the right heart contracting when the absence of a beat in the radial pulse was supposed to indicate the absence of a contraction in the left ventricle. As will be shown later, this sort of evidence is not only unreliable but actually misleading.

CHAPTER XI

Examination of the Arterial Pulse

§ **87. Superiority of the digital examination.**—In the examination of the arterial pulse, several methods may be employed, as exploration by the finger, by graphic records, and by instrumental measurement of the arterial pressure. By far the most important of these methods is the first. There is a tendency to exalt the others at the expense of the digital, but no apparatus can ever replace the trained finger. No doubt the other methods can give very definite information of a limited kind, but in diagnosing the patient's condition, they should only supplement the digital examination.

The mechanical methods can be of use, however, in enabling us to appreciate the meaning of the sensation felt by the finger, and the attempt should always be made to correlate these sensations with the results obtained by the more elaborate means.

Warning must be given against estimating the patient's condition by the study of the pulse alone ; any definite result obtained must only be employed as one of a group of symptoms on which the ultimate opinion is based.

§ **88. What is the pulse ?**—In order fully to appreciate the study of the arterial pulse, it is essential to have a proper conception of the true nature of what it is we perceive when we examine the pulse with the finger. Broadbent [136] very properly calls attention to a universal misconception of what the pulse is, and points out that it is not an expansion of the artery due to the blood discharged into the aorta. Marey [165] says that the expansion

is so slight that many physiologists have denied its existence, and he states that Poiseuille has demonstrated that in the larger arteries a slight expansion with each systole does take place. No doubt the aorta and its primary branches are somewhat dilated by the injected blood, but whatever the expansion may be in them, in the carotid and radial it must be very minute. To feel the pulse or to take a tracing, it is necessary that the artery should be flattened against the bone. Lister states that it is for this reason that surgeons operating in close proximity to a large artery may be utterly unconscious of its neighbourhood, unless they inadvertently wound it or recognize its pulsation by having compressed it against some resistant structure. The visible movements of the artery are extremely deceptive. They often give the appearance of contracting and expanding, but if the movement be critically examined, it will be found to be, in reality, a displacement of the artery. A straight artery like the carotid resembles somewhat a cord

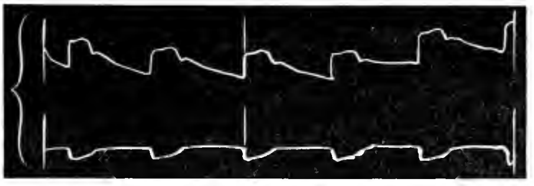

Fig. 35. The upper tracing was taken with the receiver over the carotid artery, at the same time as the lower one was taken with the receiver placed by the side of the carotid artery. The lower tracing is the inverse of the upper.

that is periodically tightened and slightly relaxed. During the systole of the ventricle, the carotid is straightened and tightened, and it becomes slightly relaxed during the ventricular diastole. In persons with thin necks this movement can be studied. If we place one receiver over the carotid and one alongside it, and have the movements properly registered, the one tracing will be found to be the exact reverse of the other (Fig. 35). If the artery expanded during the ventricular systole, it would naturally thrust out all the tissues surrounding it, and the tracing from the side would then be an exact duplicate of the one taken from the front of the artery. The movement, then, of the beating carotid is one of displacement of the whole vessel, not a dilatation and contraction of the vessel.

A similar confusion arises in studying an artery when it is tortuous. In looking at the radial when it is tortuous, one can readily imagine that the rising and falling of the artery is really a distension and contraction of the artery. But if a suitable case be taken where in the course of the tortuous artery there is a short lateral bend, the movement can be demonstrated

to be due to the displacement of the artery, and not due to expansion and contraction of the artery. If the pad of the sphygmograph spring be placed close to the artery on the concave side of the bend, and a tracing taken, it will be found that during the ventricular systole the bend is exaggerated, the artery being pushed farther away from the straight course, and during ventricular diastole the bend diminishes. If tracings be taken of the pulse in such a radial artery at the same time as the carotid pulse (Fig. 36), it will be found that the radial gives an inverted tracing comparable to that in the preceding figure. If the visible movement were due to the expansion and contraction of the artery, the lever would, on the contrary, rise during systole and fall during diastole, as in an ordinary sphygmogram.

What we recognize then as the pulse is the sudden increase of pressure within the artery pressing against our finger when we compress the artery. With the cessation of the ventricular systole the resistance to our finger steadily diminishes until the next ventricular systole suddenly rises.

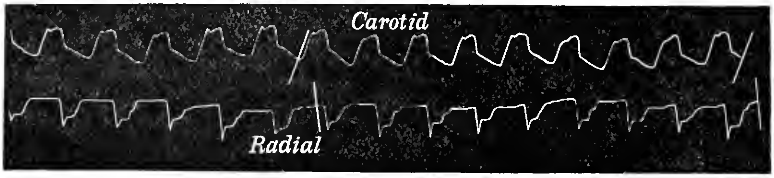

FIG. 36. Simultaneous tracings of the carotid and radial pulses. The radial tracing was taken by placing the pad of the sphygmograph by the concave side of a bend of the tortuous radial artery. During systole the artery receded from the sphygmograph and returned during diastole, and hence the tracing obtained is an 'inverted sphygmogram'.

Broadbent [136] uses the following apposite illustration: 'Such a pulsation can be felt on a large scale by placing the foot on the inelastic leather hose of a fire-engine in action, in which there can be no expansion.'

To speak of the pulse as being the expansion and contraction of the arterial walls, or 'the swinging backwards and forwards of the arterial wall', is not only to use language of exaggeration, but to convey a totally erroneous conception of what the pulse really is.

§ 89. **Inspection of the arteries.**—Inspection of the arteries reveals in health but little movement. Conditions giving rise to forcible action of the left ventricle may render the pulse visible in some of the superficial arteries. Exertion, excitement, or the febrile state may induce visible beating of the carotids, while this is a marked feature when the arteries are tortuous and atheromatous, and in such a disease as exophthalmic goitre. In free aortic regurgitation, not only is there marked pulsation of the carotids, but pulsation is visible in numerous superficial arteries in various situations.

The tortuous character of superficial arteries is visible in arterial degeneration.

§ 90. **Digital examination of the arteries.**—It is usual in the routine examination of the pulse to place two or three finger tips on the radial artery near the wrist. The fingers are laid on the artery, and moved upwards and downwards and across the artery, at first gently, and then with more pressure. By this procedure a knowledge of the size of the artery and the conditions of its walls is acquired. Steady pressure being applied in order to obliterate the pulse, the force required to attain this gives an idea of the arterial pressure, and of the character of each individual pulse-wave. It is a good thing to practise the digital examination of the pulse with sphygmographic tracings taken at the same time. By this means, the character of the pulse will be better appreciated by the finger.

The condition of the walls.—We recognize the yielding nature of the arterial coats in healthy arteries. In degeneration of the coats the arterial walls may be universally thickened, or contain bead-like patches of induration as in atheroma, or the artery may have become a rigid tube as in calcareous degeneration.

The size of the artery.—The variations in size depend entirely upon the degree of relaxation of the muscular coat of the artery. A large artery is not necessarily significant of a strong pulse, nor a small artery of a weak pulse. An increase in the size of the artery frequently implies diminished opposition to the work of the heart. The size of the artery can sometimes be readily appreciated by lightly rolling it under the fingers. At other times it can only be detected when the pulse is elicited by firm pressure at the place where we expect to find it. This difficulty may occur where there is a good-sized artery embedded in a fat, well-padded wrist, or where the artery is small and contracted. On the subsidence of a fever, a notable diminution in the size of the artery can often be readily recognized.

The arterial pressure.—The trained finger is as yet the best guide we have in judging the pressure within an artery. The knowledge necessary to determine what is normal and what is abnormal, can only be acquired by the constant study of the pulse. The finger tips become so educated in course of time that we readily appreciate the sensation conveyed in compressing an artery (see Chapter XII).

The pulse-rate.—The reckoning of the pulse-rate should be made at a late stage in the examination. When abnormally quick, it should be again counted when the patient has regained his composure. It is best enumerated in two separate half-minutes, to ascertain if the heart is acting quite steadily. In children, unless asleep, abnormal frequency is often

very unreliable as a guide, as the presence of the doctor often keeps up a continued excitation of the heart. While the rate of the pulse normally indicates the number of the contractions of the left ventricle, it sometimes happens that these are so weak that some of the pulse-waves are not perceptible to the fingers. In such cases the pulse is usually slow or irregular in rhythm. To appreciate the significance of the pulse-rate, due regard should be paid to the age and idiosyncrasies of the patient, and to the ailment from which he suffers.

The size of the pulse-wave.—The trained finger can recognize a great variety in the apparent volume of the wave itself. Some waves seem to roll up under the finger, passing gradually away, while others pass quickly, giving a mere flick to the finger.

The impact of the pulse-wave on the finger.—This may be quick and abrupt, and the pulse-wave quickly disappear (pulsus celer), or the impact may approach the finger gradually and gradually subside (pulsus tardus). Although the pulse-wave occupies such a short space of time, yet the sensitive finger readily recognizes these different features.

The rhythm of the pulse.—The beats usually follow one another at regular intervals, and should be of equal strength. The divergencies from the normal rhythm are numerous, and the usual terms employed to distinguish them are, in my opinion, both unsatisfactory and misleading, but this subject is fully entered into later. In estimating the rhythm of the pulse, one's whole attention should be concentrated upon the observation. If one does not exclude other thoughts from the mind, a variation in the pulse rate and strength may apparently be felt. This is due to a failure to appreciate the pulse during a remission of the attention. I have not only been conscious of this myself, but in cases where it was important to note the fact, as in pneumonia, I have found my colleagues describing irregularity as being present, when careful examination revealed a perfectly regular pulse.

The two radial pulses compared.—Finally, the two radial pulses should be compared, and any difference in the character of the beats noted. A difference in the strength of the two pulses may be due either to an abnormal distribution of the arteries on one side, or to an interference with the lumen of a vessel on one side. A difference in the character of the pulse usually occurs only in the latter case. The two most frequent conditions altering the character of the pulse on one side, are the presence of an aneurysm or of an atheromatous plate, diminishing the lumen of the vessel, on the proximal side of the place where the pulse is examined.

§ 91. **The value of a sphygmogram.**—Although the sphygmogram

represents the variations in arterial pressure, and although it can give information in this respect, yet there are so many sources of error, that it cannot be trusted implicitly. Its greatest service is in giving an accurate record of the movements of the left ventricle. However eloquent may be the words of a writer, he cannot in a page convey as clear an idea of the rhythm of a heart as a simple pulse-tracing, and if writers had given us more pulse-tracings their works would have been greatly enhanced in value. It is because it gives us a permanent and accurate record, that a tracing of the arterial pulse is of such great value. When we seek to find the nature of any movement of the circulation by recording it graphically, the arterial pulse is the best and most useful standard by which we can find its position in the cardiac cycle, as will be shown later.

§ 92. **Definition of a sphygmogram.**—When the spring of a sphygmograph is so accurately adjusted on an artery that it does not obliterate the artery when the arterial pressure is at the lowest, and still slightly compresses the artery when the arterial pressure is at the highest, the spring will oscillate with each variation of pressure within the vessel. This oscillation being communicated to the lever and recorded on the tracing-paper gives us a series of wavy lines, which represent the variations of the pressure within the artery. A sphygmogram may therefore be defined as a diagrammatic representation of the variations of pressure within an artery. If we knew exactly the amount of pressure exercised by the spring, we should be able to obtain the value of each movement. But the possibilities of error are so numerous that it is useless to draw conclusions from the amount of pressure supposed to be exercised. From the examination of a tracing we obtain information on three different points: first, concerning the rate and rhythm of the heart's action; second, concerning the sequence of certain events occurring in a cardiac revolution; third, concerning the character of the blood-pressure within the artery.

§ 93. **Events occurring during a cardiac revolution revealed by the sphygmogram.** (*a*) *The systolic period.*—If we take a sphygmogram we can divide the cardiac cycle into two periods: one (*E*, Fig. 37) during which the aortic valves are open and the ventricle pours its contents into the aorta, and another (*G*, Fig. 37) during which the aortic valves are closed and the ventricle is in diastole. For the sake of convenience in describing sphygmograms these two periods will be referred to as the systolic and diastolic periods, although in the space *G* the presphygmic and postsphygmic periods of the ventricular systole are included (Fig. 43). The character of the systolic portion varies very much in different individuals. These variations depend mainly on the amount of resistance offered by the arteries to the ventricular

systole. In such a tracing as Fig. 37 there is first an abrupt rise (p), then a fall followed by a continuation of the wave (s) at about the same level. This period is usually described as being divided into two, the abrupt rise being spoken of as the primary or percussion wave, and the latter portion as the tidal or predicrotic wave (the papillary wave and outflow remainder wave of Roy and Adami). This division has led to the idea that these represent two different events in the pulse itself. As a matter of fact, the

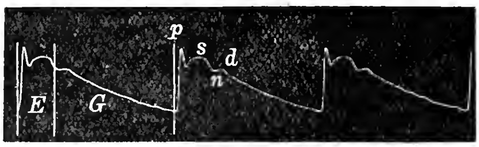

FIG. 37. Sphygmogram of the radial pulse. The space E is the period of ventricular systole when the aortic valves are open; the space G the period of ventricular diastole; s is the pulse-wave due to the ventricular systole; n the aortic notch; d the dicrotic wave; and p a wave due to instrumental defect.

abrupt rise p above the level of the wave s is due to instrumental defect, and the whole period E is occupied by the ventricular pressure forcing blood into the arterial system, and corresponds with the period E in Fig. 43. In cases where the arterial pressure is low relatively to the strength of the ventricular systole, these two waves are so blended together that so-called percussion and tidal waves can no longer be differentiated (Fig. 38). The whole of this period E in the tracing will hereafter be referred to as the systolic period, and the wave s as the systolic wave, as it represents the

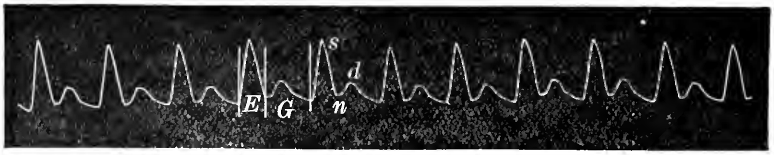

FIG. 38. The letters have the same significance as in Fig. 37.

period of ventricular systole when the ventricle and arterial system are in free communication.

(*b*) *The diastolic period.*—With the closure of the aortic valves, the arterial pressure falls rapidly to the bottom of the aortic notch n (Figs. 37 and 38). In the tracings this is seen to be at the beginning of the diastolic period. This fall is interrupted by a distinct rise in the pressure represented by the dicrotic wave d. There has been a good deal of discussion concerning the cause of the dicrotic wave. The following explanation seems to me the most probable. The semilunar valves are so delicately constructed that

they readily respond when the pressure on one side rises above that on the other. As soon as the aortic pressure rises above the ventricular the valves close. At the moment this happens the valves are supported by the hard, contracted ventricular walls. The withdrawal of the support by the sudden relaxation of these walls will tend to produce a negative pressure wave in the arterial system. But this negative wave is stopped by the sudden stretching of the aortic valves, which, on losing their firm support, have now

FIG. 39. A strong-beating ventricle has jerked the lever high above the true systolic wave, and the falling lever has made an artificial notch on the systolic wave *s*. The true pulse curve is probably represented in the dotted tracing.

themselves to bear the resistance of the arterial pressure. This sudden checking of the negative wave starts a second positive wave, which is propagated through the arterial system as the dicrotic wave. After the dicrotic wave the arterial pressure-curve gradually falls. Occasionally there are slight waves in the fall, but these are of doubtful import.

§ 94. **Features of the sphygmogram due to instrumental defect.**— In the study of sphygmographic tracings one has always to bear in mind that

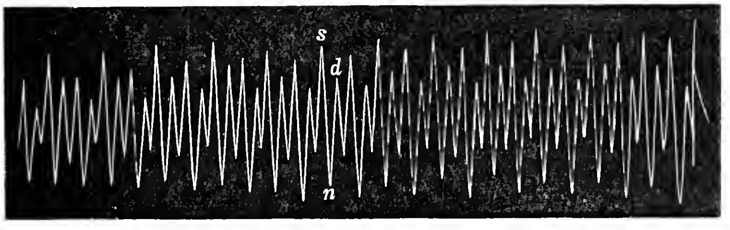

FIG. 40. The forcible changes in pressure have exaggerated the height and depth of all the waves.

certain features may be due to the instrument itself. Speaking generally, these instrumental features occur where there are sudden and forcible changes in the arterial pressure. The most frequent of these is the jerking up of the spring by the systolic wave itself. Then the next most frequent is the formation of a notch on the tracing of the systolic wave, due to the sudden fall of the spring after being jerked high up, as in Fig. 39. Occasionally one finds the aortic notch artificially deepened by the sudden lowering of the pressure, as in Fig. 40.

CHAPTER XII

ARTERIAL PRESSURE

§ **95. The cause of arterial pressure.**—When the left ventricle contracts it drives the blood into the arterial system. The escape through the arterioles and capillaries is retarded, so that the blood continues to flow after the ventricle has ceased to contract. As a consequence of this, the arteries are slightly distended during ventricular systole, and their elastic coats compress the column of blood within them after the ventricular systole is over, and thus maintain a degree of arterial pressure during the period in which the ventricle is not acting. The ventricular force is thus stored up by the distension of the elastic coats of the arteries, and liberated during the ventricular diastole.

The chief factors therefore concerned in the maintenance of arterial pressure are the ventricular systole, the peripheral resistance, and the elastic recoil of the arteries. The viscosity of the blood is also a factor in the raising of the arterial pressure.

§ **96. Methods of measuring the blood-pressure.**—Of late years many instruments have been devised to measure the arterial pressure. The majority are constructed on the principle of compressing the brachial artery with an air-bag embracing the upper arm. Air is pumped into the bag, and its pressure is measured by a mercury manometer in connexion with it. When the pressure is raised sufficiently to obliterate the radial pulse, we obtain the only really trustworthy standard, and it is this I refer to hereafter as ' arterial or blood pressure '. Attempts have been made to estimate the systolic, mean, and diastolic pressures by observing or recording the movements communicated to the column of mercury by the compressed artery. It is found that, during the gradual compression of the artery, oscillations due to the pulse beat occur in the mercury. These oscillations begin, gradually reach a maximum, and gradually decrease as the pressure

is raised or lowered. Far-reaching deductions have been drawn from the changes in these oscillations. I think, if the cause of these oscillations be properly appreciated, no safe deductions can be drawn as to what is called the systolic, mean, or diastolic pressures. When the pressure within the air-bag corresponds to that of the artery, the arterial pulse communicates its impact to the air in the bag, and thus induces the oscillation of the mercurial column. The size of these oscillations depends on the amount of movement of the arterial wall. Let the lines, *A*, *B*, Fig. 41, represent the walls of the artery, and for simplicity assume that the pressure is applied to one side only of the vessel. It is found that a pressure of 160 millimetres obliterates the arterial pulse, the wall *A* being flattened down on *B* with such force that no blood passes through, and there is no oscillation of the mercurial

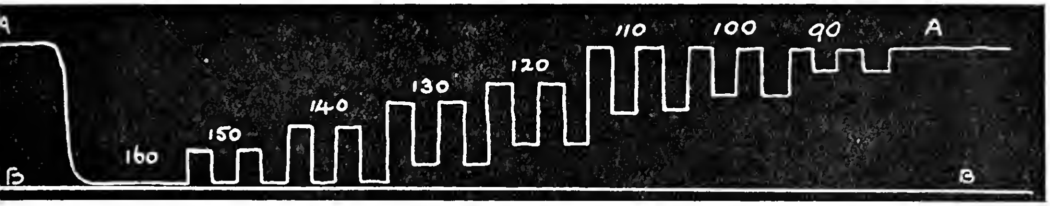

FIG. 41. Diagram to show the nature of the oscillations of the mercurial column in estimating the blood-pressure. *A* and *B* represent the walls of the brachial artery. At 160 the lumen of the artery is obliterated by the pressure of an air-bag embracing the upper arm. As the pressure is gradually lowered in the bag, each beat of the artery produces a movement of the mercury in the manometer, the movement being represented by the elevations in the diagram. With diminution of pressure there is at first a gradual increase in the extent of the movement followed by a gradual decrease until the pressure in the air-bag ceases to compress the artery. From this it is shown that there is no definite period which can be said to correspond to the systolic, diastolic, or mean blood-pressure.

column. When the pressure is lowered to 150, a small wave escapes during systole which raises the wall to a slight extent, and gives rise to a small oscillation. At 140 a larger wave escapes, but the pressure of 140 is still sufficient during a portion of diastole to flatten the wall *A* against *B*. At 130 the pressure no longer flattens the wall *A* against *B*, but the flow escapes during diastole, and the oscillation reaches its maximum. With the gradual lowering of the pressure, the maximum oscillation persists. At 110 the artery is fully distended during systole, and only partially compressed during diastole, but the pulsation is still maximal. As the pressure falls there is a decrease in the compression of the artery and a corresponding decrease in the size of the oscillation, until the pressure fails to compress the artery sufficiently to produce any movement. From this way of looking at the matter, it will be realized that at no definite period

can we tell when the record represents a systolic, a mean, or a diastolic pressure, and that the maximal oscillation may last through a considerable range of pressure with nothing to guide one to the moment when any definite pressure could be ascertained. The diagram may not represent the condition of affairs in every case, and a limited usefulness may possibly be found in noticing the period of maximal oscillation and its decline, but so far, I think, no very reliable conclusion can be drawn.

The force required to obliterate the pulsation in the radial artery is fairly easily ascertained, and from it certain limited inferences may be drawn. It is doubtful if it represents the actual arterial pressure within the artery, for an escape can take place imperceptible to the finger, and certain external conditions may affect the pressure. It is usually assumed, for instance, that the arterial wall and its coverings offer such a slight resistance as to be negligible. Russell [427], on the other hand, asserts that the thickening or contraction of the artery may have a very considerable effect, and that thick, sclerosed, and contracted arteries may offer such resistance that a considerable proportion of the pressure may be spent in overcoming it. Oliver [82] has shown that the pressure obtained by instruments may vary in different arteries in the same individual, while L. Hill's observations [71] show that the arterial wall can have only a very slight influence in the instrumental observation of the blood-pressure.

There are a great many different instruments devised to take the arterial pressure. Most of them are somewhat cumbrous, and some patients resent the disagreeable sensation produced by compressing the upper arm. Hence these methods are not likely to receive that general application which the need for ascertaining the blood-pressure requires. L. Hill has recently invented an extremely simple and practical method. In place of a cuff surrounding the upper arm, he uses a small bag which is compressed over the radial artery until the pulse disappears below the place of compression. The bag is connected with a very simple manometer, which can be carried in the waistcoat pocket.

Erlanger [64] and Gibson [68] have invented methods for graphically recording the blood-pressure. The tracings from Gibson's apparatus seem very instructive, but I have no experience in the use of his apparatus.

A great deal has been written about blood-pressure and its estimation in practical medicine, but it must be confessed that much of it has been of little practical value, and much careful observation, extending over many years, on individual patients, will be necessary before any sure and certain result is obtained. The remarks I make on the subject are based on the examination of numerous patients in the endeavour to ascertain some sure

foundation on which to base the application of the method in the clinical examination of heart affection.

§ 97. **Increased blood-pressure.**—In many cases, one can corroborate by instrumental observation the knowledge previously acquired by the finger, that the pressure in the arteries increases in certain diseased conditions, as Bright's disease, and with advancing years. As the increased pressure of advancing years is associated with arterial changes, the question of cause and effect is a very difficult one to solve. On the one hand, the changes in the blood-vessels undoubtedly tend to raise the blood-pressure, while it is contended that these are induced by the blood itself containing ingredients that provoke a contraction of the arterioles, in consequence of which the muscular coat hypertrophies. A rise in pressure seems to induce atheromatous degeneration, and this in turn causes a rise in pressure, and thus a vicious circle may be formed. The fact is undoubted, that arterial changes and high blood-pressure are very frequent phenomena in advanced life. The changes are so insidious that they rarely come under consideration until they are well established. One may infer, indeed, that such changes are occurring in middle life, when one notices a tortuous temporal artery, but it is rare that the condition gives any cause for anxiety until it is well established.

It is a mistake, and one made not infrequently, to begin treating the high blood-pressure as if it were a disease. Happily the efforts employed to reduce the blood-pressure are usually of little value. In order to appreciate the meaning of high blood-pressure, it is well to consider the condition associated with its production, for I think it has a significance beyond that of being a manifestation of disease.

I have already pointed out that the arterial pressure is maintained chiefly by the force of the left ventricle, the peripheral resistance, and the elastic recoil of the arteries. The necessity for the pressure is the regular and equable supply of blood to the organs and tissues. Between the heart and tissues there is an intimate association, whereby the supply to the tissues and organs is moderated by their requirements—the heart beating more forcibly and more rapidly, and the peripheral resistance diminishing when there is an urgent need, by the exercise of the functions of the organs. With advancing age, three great changes occur in the blood-vessels. The elasticity of the arteries diminishes. The result of this is that there is no longer the same equable maintenance of the pressure during diastole. The arteries approximate the condition of rigid tubes where the force exerted by the left ventricle is not sufficiently stored up in the elastic coats, to be liberated during diastole. The loss of this assistance necessitates increased

force of the ventricular contraction, and therefore an increase of the pressure during ventricular systole. In the arterioles there may be an increase of the muscular tissue, and this implies an increase in functional activity with an increase of the peripheral resistance. These are the two factors that are generally assumed to be the cause of increased blood-pressure, but there is a third which has not received that consideration to which it is entitled, namely, the diminution of the capillary field. This can be recognized in various ways, as, for instance, the thinning and wasting of the skin and subcutaneous tissues, and by the absence of oozing in surgical operations. The manner in which it raises the blood-pressure is simply by narrowing the passage of outflow (see § 221).

§ 98. **Hyperpiesis.**—In the routine examination of patients we meet occasionally with some, usually middle-aged, sometimes young, who show considerable fluctuations in blood-pressure. Periods of high arterial pressure (hyperpiesis of Clifford Allbutt [58]) may be associated with some discomfort, as mental dullness, headache, &c. These periods can be cut short by a smart purge, bodily exercise, &c., or they disappear from no ascertainable cause. It is possible the periods of high arterial pressure are due to faulty metabolism, but they will be found to recur in spite of the greatest care in diet. It is said by some that these periods of high blood-pressure are the cause of arterial degeneration, but, with an imperfect knowledge of all the factors we are not in a position to decide.

§ 99. **Effect on the heart of increased peripheral resistance.**—I have mentioned that the connexion between the heart and the tissues is so intimate that the demand from the tissues is responded to by stronger contraction of the heart. When, therefore, one or all of these causes increasing the peripheral resistance are in action, the heart, in order to supply the tissues with blood, has to exert more force in its contraction. It accommodates itself to its ever-increasing burden by calling upon its reserve, and the only evidence that it has more to do is in the limitation of its field of response. One can almost say, when an individual realizes the fact that a hill is not climbed with the ease and comfort with which it used to be done, that already the heart is meeting an increased peripheral resistance, and there is already a slight exhaustion of its reserve force. This, as we know, is a very gradual and long-continued process, beginning insidiously in the fourth decade of life, and coinciding with the time at which athletes abandon the exercises that call for long and severe exertion.

As the changes that increase the peripheral resistance tend slowly but surely to advance, the work of the heart becomes ever greater, the field of response becomes more limited, till finally the patient's attention is called

by some disagreeable sensation to the fact of the great limitation, and so we get heart failure. The peculiar feature of the heart failure that arises in this way is, that the function of the heart that has most work to do usually fails, namely, the function of contractility, so that it is in these cases of high arterial pressure that we meet the most typical symptoms associated with failure of the function of contractility.

But this is not all. The changes that have taken place in the arterial walls in the periphery have at the same time been affecting the arteries of the heart, with the result that the muscle-fibres are imperfectly nourished and degenerate. It is in these cases of long-standing high blood-pressure that we find the most striking evidence of degenerative changes in the heart muscle associated with arterial degeneration, and it is wonderful how long a heart extensively degenerated can maintain a high blood-pressure.

Such cases of degenerated arteries and long-continued high blood-pressure end by a degenerated artery giving way and causing cerebral apoplexy, or by failure of the heart. The latter may come about in various ways: by a sudden change in the inception of the heart's contraction—for it is in these cases we frequently find the extra-systole and nodal rhythm—by gradual exhaustion of the contractility, often with angina pectoris, and on rare occasions by rupture of the heart. There may occur a somewhat sudden fall in the arterial pressure from dilatation of the heart. When this occurs there is a sudden change in the character of the symptoms which I have given in some detail in the chapter on dilatation of the heart (Chapter XXIII).

§ 100. **Increased arterial pressure and heart failure.**—From such considerations it will be realized that we may have heart failure without fall of blood-pressure, and this consideration brings clearly into view the fact that heart failure in these cases is primarily a matter of exhaustion of reserve force. In cases of valvular disease, as H. Starling [87] points out, there may be marked failure of the heart with little or no fall in blood-pressure, and recovery may ensue with little or no rise in blood-pressure. It will be found, however, in these cases that the heart failure is not associated with a loss of the function of tonicity. When dilatation occurs, then there is usually a fall of blood-pressure, and recovery, when it takes place, is accompanied by a rise of blood-pressure.

§ 101. **Treatment of high arterial pressure.**—It is an almost universal custom to treat the signs of heart failure with 'cardiac tonics'. By cardiac tonics are usually meant drugs or methods that are supposed to raise the blood-pressure. Hence, when any disorder that implies inefficient action of the heart is met, such drugs as digitalis, or some methods of exercise and baths supposed to raise the blood-pressure, are resorted to. It is manifest,

from the consideration of the causes that induce high blood-pressure, that this treatment is incorrect. Happily the methods and drugs have far less effect than is supposed, and as rest is usually enjoined at the same time the good results are due more to unconscious adoption of the best principle of treatment than to the efforts that are supposed to be remedial. Manifestly, in heart failure induced or aggravated by high pressure, the best line of treatment is to ease the load and give the heart rest, to regain some store of reserve force. Now there is a tendency to rush to the opposite extreme, and to endeavour to reduce the blood-pressure by administering drugs of the vaso-dilator class. Luckily, the administration of these drugs is of little effect, and little or no permanent lowering can be obtained by their use. It is manifest that with changed arteries, and a diminished capillary outflow through obliteration of the capillary vessels, a high blood-pressure is necessary to supply the organism with blood. If it were possible to reduce the blood-pressure permanently in a man who for years had a blood-pressure of 180–200 mm., the result would be impaired nutrition of the organism. If the final breakdown of these patients be watched, it will not infrequently be found that the blood-pressure does fall to 150 or 140 mm. Hg., and the result is at once the appearance of the signs of extreme heart failure —dropsy, enlarged liver, oedema of the lungs, &c. So serious is the significance of a fall of blood-pressure in patients with cardio-sclerosis, even with attacks of angina pectoris, that the persistent fall of pressure is an evidence of the final exhaustion of the heart, though the anginal attacks may cease.

The principle I pursue with most success is to place the patient under conditions that give the heart less work to do, carefully avoiding 'cardiac tonics' and 'vaso-dilators', restricting the diet, evacuating the bowels, and permitting such exercise as the patient can undertake without distress, according to the lines laid down in the chapter on treatment.

§ 102. **Diminished arterial pressure.**—The question of low pressure and its significance is an extremely puzzling one. Here again one meets the confident assertion, 'In hypotonic conditions give cardiac stimulants or vaso-constrictors.' The matter is, however, far more complicated, and I am only dimly groping after the true principles of treatment in these cases. In dilatation of the heart, as best seen in mitral disease, the fall of pressure may be considerable, and the administration of drugs of the digitalis group has undoubtedly a most beneficial effect in improving the patient's condition, and in raising the blood-pressure. But this is due to the special effect that digitalis has upon the function of tonicity, both in the heart and the arterial muscle-fibres. But in low pressure associated with other conditions the drug is often of little avail.

CHAPTER XIII

The Venous Pulse

§ 103. **What the venous pulse shows.**—The consideration of the circulation has so far been mainly concerned with the effects of the contraction of the left ventricle. When the apex beat is studied, or the characters of the arterial pulse analysed, our purview is limited almost entirely to the doings of the left ventricle. The arterial pulse indeed gives us a direct knowledge of the left ventricle's action during but a portion of the cardiac cycle, namely, during the period when the aortic valves are open. When they are closed, we are no longer directly cognizant of what is happening in the left ventricle. We come now to the study of a subject which gives far more information of what is actually going on within the chambers of the heart. In the venous pulse we have often the direct means of observing the effects of the systole and diastole of the right auricle, and of the systole and diastole of the right ventricle. The venous pulse therefore presents a greater variety of features, and may manifest variations due to disease which the study of the arterial pulse fails to reveal.

§ 104. **Inspection of the jugular pulse.**—In examining a patient for pulsation in the jugular vein, it is generally best that he should lie down, though in some rare cases, where the veins are greatly distended, the pulsation can only be recognized when the patient sits up. The pulsation is most commonly limited to the internal jugular veins, and these veins, lying

alongside the carotid arteries, are never visible, being covered at the root of the neck not only by the skin and sterno-mastoid muscle, but by a variable quantity of adipose tissue ; one therefore recognizes the venous pulse only by the character of the movements communicated to the structures covering the vein. In that form of the venous pulse in which the principal wave is due to the auricular systole, the sudden collapse of the tissues covering the vein is more striking than the protrusion. If one further carefully times this collapse, it will be found to be synchronous with the arterial pulse. The pulse in the internal jugular vein is often mistaken, even by experienced observers, for ‘ beating of the carotids ’. But the carotid pulse is always abrupt and sudden in its protrusion of the covering tissues, and gradual in the shrinking. Furthermore, when one finds a small radial pulse and a large pulsation in the neck, one may safely conclude that the neck pulsation cannot be carotid, unless under very exceptional circumstances (as aneurysm). When the pulsation is in the more superficial veins, as the external jugular, facial, or superficial thoracic veins, the collapse of the vein synchronous with the carotid pulse is usually easy of recognition. In another form of venous pulse, where the pulsation is due to the ventricular systole, the engorgement of the veins is usually so great, the arterial pulse so small, and the cardiac mischief so evident, that the recognition of the venous pulse is comparatively easy.

§ **105. Methods of recording the jugular pulse.**—Usually the movements of the vein are best recorded with the patient lying down, the shoulders slightly raised, the head comfortably supported by a pillow, and turned slightly to the right in order to relax the right sterno-mastoid muscle. The receiver (*E*, Figs. 13, p. 68, and 18, p. 73) is placed over the jugular bulb immediately above the inner end of the right clavicle, with just sufficient pressure to shut off the interior of the receiver from the outer air. One may have to shift the receiver about to get the best movement. The relation of the jugular bulb to the surrounding structures is shown in Fig. 42, where the circle above the clavicle indicates the position of the receiver.

Sometimes better tracings are got higher up in the neck or from the left side. In great engorgement of the veins it may be possible only to get a tracing when the patient is sitting up. The continued action of the sterno-mastoid in laboured breathing may prevent a tracing of the jugular pulse being obtained.

§ **106. The recognition of the events in a jugular pulse.**—There is still much that is obscure about some of the details of the venous pulse, and several of these are still the subject of controversy. In the following interpretation I deal with the salient points which have thrown most light

upon the obscure features of the heart's action. The movements of the venous pulse are usually more numerous than those of the arterial, and in the tracings a number of waves are present. As each of these indicates a rise of pressure in the veins, the tracing can only be properly interpreted when the force producing each rise of pressure is known; and for this purpose the time of appearance of each wave in the cardiac cycle must be

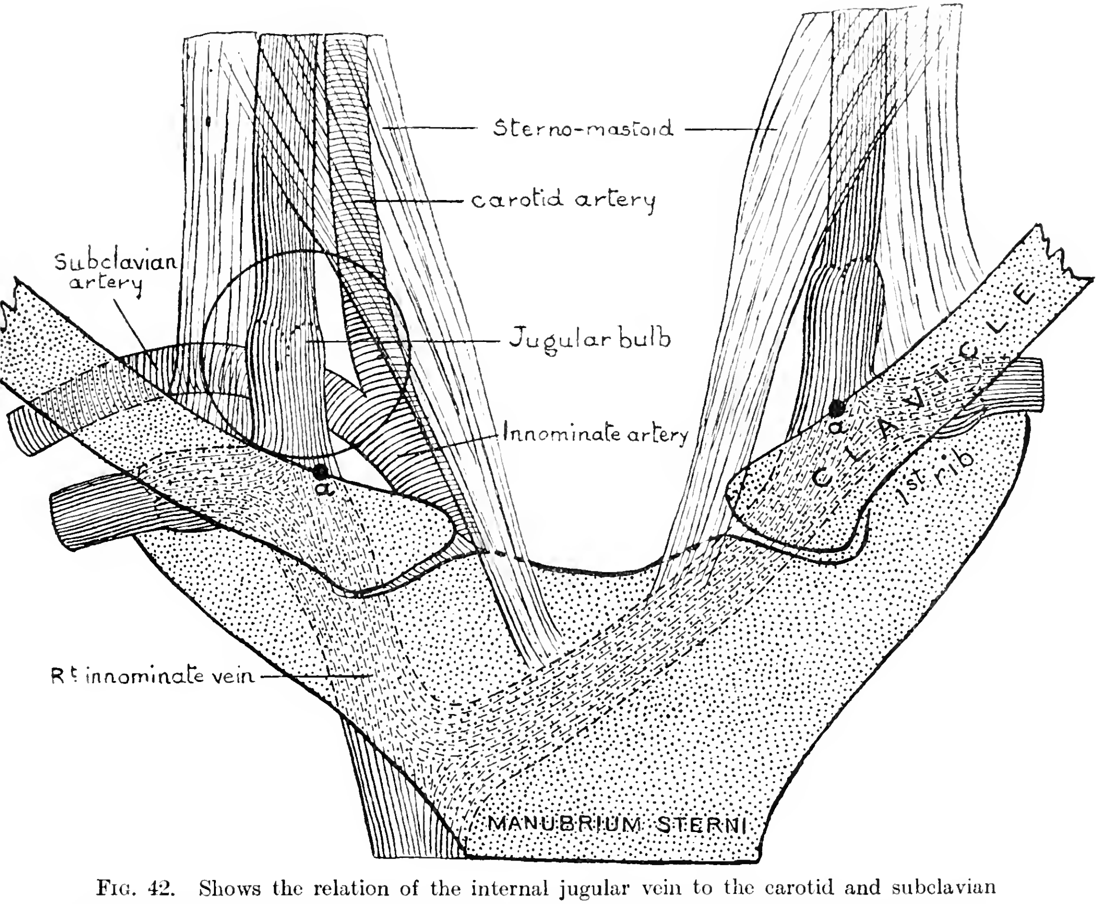

FIG. 42. Shows the relation of the internal jugular vein to the carotid and subclavian arteries, and to the sterno-mastoid muscle. The circle represents the position of the receiver in taking a tracing. The spot at *a* is one inch from the internal end of the clavicle. (Keith.)

established. This is done by taking tracings of the venous pulse, at the same time as some movement whose position in the cardiac cycle is definite, and the arterial pulse, carotid or radial, is the most reliable. The apex beat is often useful and convenient, but care is necessary in the employment of the apex beat, as has already been pointed out (§ 84).

§ 107. **Description of the events in a cardiac cycle.**—In the diagram (Fig. 43) there is represented a series of movements due to various forces

that occur during one cardiac cycle. If a wave be found in the vein, and if its time of occurrence be ascertained by referring to the place it would occupy in this diagram, we can usually find its cause by noting what force is operative at that period. It must be added that while this diagram represents with fair accuracy the chief events in a cardiac revolution, it is not asserted that it is correct in every detail. Authorities are not quite

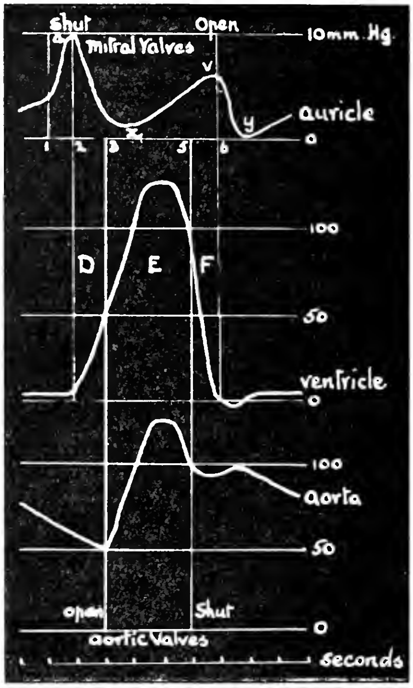

Fig. 43. Semi-diagrammatic representation of the auricular, ventricular, and aortic pressures during one cardiac revolution. *D*, the presphygmic period of the ventricular systole; *E*, the sphygmic or pulse period; *F*, the postsphygmic period. The figures 1, 2, 3, 5, and 6 have the same significance as those in Fig. 46. The divisions on the bottom line represent tenths of a second. (After Frey.)

agreed on several small points, but it is sufficient for the purpose I have in view.

What we have here presented are, the curves representing the variations of (1) the pressure within the auricle; (2) the pressure within the ventricle; (3) the pressure within the aorta. The spaces embraced by the perpendicular lines represent respectively the time during which the semilunar valves are open (*E*) and the auriculo-ventricular valves are shut (*D*, *E*, *F*). I would

direct attention to the presphygmic period D, when the ventricular pressure is rising, but has not yet opened the aortic valves; and to the postsphygmic period F, where the ventricular pressure is falling after the closure of the aortic valves. The curves indicating the pressures are approximately correct, but are utilized here to show the periods when variations take place in the pressure. Though the events in the diagram represent what happens in the left side of the heart, there can be no doubt that the changes on the right side are of the same character.

§ 108. **The causes of the variation of pressure in the auricle and in the jugular vein.**—The auricular pressure in Fig. 43 shows a series of rises and falls, and these correspond to those in a venous pulse (Fig. 44), The forces operative in producing the variations in the auricular pressure

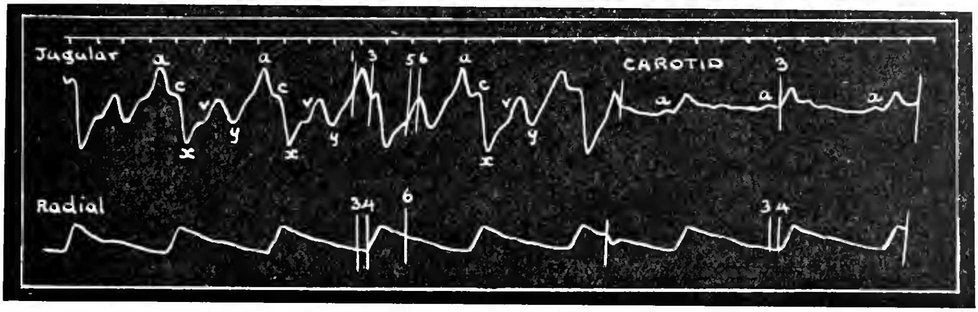

FIG. 44. Simultaneous tracings of the jugular and radial pulses and of the carotid and radial. The elevations a and v, and falls x and y in the jugular tracing correspond to those in the auricular pressure-curve in Fig. 43.

are also acting in producing the jugular pulse. (The curves of auricular pressure by different physiologists are very perplexing, some getting a rise of varying duration during ventricular systole. I select Frey's as the simplest and probably the truest.)

The auricular wave (a) *and fall* (x).—The rise a in the jugular pulse, Fig. 44, corresponds with the first abrupt rise a in the auricular pressure-curve, Fig. 43, and both are due to the systole of the auricle. Neglecting the wave c for discussion later, the fall x in the jugular pulse corresponds to the fall x in the auricular pressure-curve, and occurs at the time the ventricle is in systole. The fall is due to three factors: (1) the relaxation of the auricle after its systole; (2) the dragging down of the a.-v. septum by the ventricular muscle, enlarging the auricular cavity as described in § 20, and Fig. 3; (3) the diminished intrathoracic pressure in consequence of the expulsion from the chest of the contents of the left ventricle. When there is a delay in the ventricular contraction, the factors may be separated as in

Fig. 112, p. 177, where x is due to the first factor, and x' to the second and third factors.

The ventricular wave (v).—The rise v, after the fall x (Fig. 44), is due to the storing of blood in the auricle during the time of the ventricular systole, and corresponds with the second rise (v) in auricular pressure-curve in Fig. 43. The termination of this rise in both figures is sudden, and due to the opening of the auriculo-ventricular valves. *While the beginning of this rise is very variable, its termination is one of the most certain periods in the cardiac cycle, indicating as it does the time of the opening of the tricuspid valves.* The variableness in its beginning is due to the fact that it owes its origin to the quantity of blood stored in the auricle during ventricular systole, and this varies in individual cases, and also in the same individual,

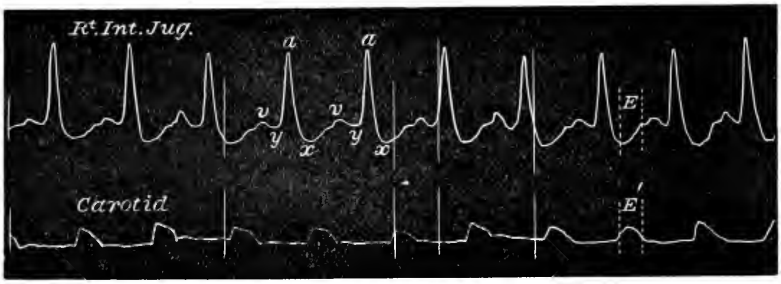

FIG. 45. Simultaneous tracings of the jugular and carotid pulses, showing that the waves in the jugular correspond in time with the waves of increased auricular pressure in Fig. 43. In this patient the tricuspid valves were partially destroyed and the orifice therefore markedly incompetent, so that there was free tricuspid regurgitation. The wave v is therefore partly due to blood regurgitating from the right ventricle.

with exertion and respiration. The blood comes mainly from the periphery, pouring into the auricles through the veins. When the auricle becomes filled, the surplus distends the superior vena cava and jugular, and hence appears in the tracing as a wave. Another source is sometimes regurgitation through the tricuspid orifice (see § 212). It is necessary to bear this in mind, as the failure to recognize how tricuspid regurgitation would be manifested in the venous tracing has led to a total misconception of the meaning and nature of the ventricular form of the venous pulse. It has been assumed that in tricuspid regurgitation the blood sent back into the veins would appear in the jugular at the same time as in the carotid. In this assumption the effects of a dilating auricle between the ventricle and veins has been overlooked; as a matter of fact, what happens is merely an increase in the amount of blood accumulating in the auricle during the ventricular systole, and this causes the appearance of the wave v to be somewhat premature. Thus, for instance, Fig. 45 is from a case where there was a damaged tricuspid valve,

so that regurgitation took place, and the wave v is seen to be of small size, beginning early in the time of the ventricular systole (period E).

I call this wave (v) the ventricular wave, because of its association with the systole of the right ventricle. Thus the termination of the wave is due to the relaxation of the right ventricle and opening of the tricuspid valves ; it is often made up of blood sent back through the incompetent tricuspid orifice by the systole of the right ventricle ; though this wave may be small and of brief duration in the auricular form of the venous pulse, it becomes increased in size and the main or only wave in the ventricular form of the venous pulse.

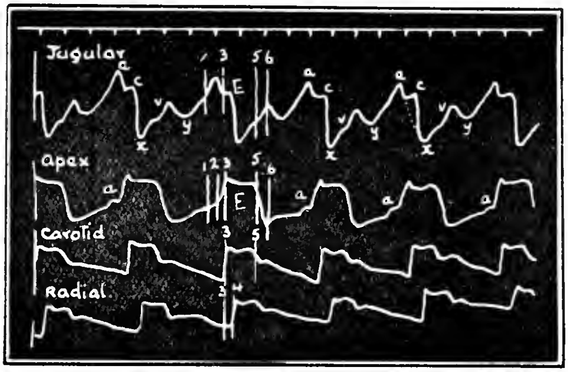

Fig. 46. Tracings of the jugular pulse, apex beat, carotid and radial pulses. The perpendicular lines represent the time of the following events : 1, the beginning of the auricular systole ; 2, the beginning of ventricular systole ; 3, the appearance of the pulse in the carotid ; 4, the appearance of the pulse in the radial ; 5, the closing of the semilunar valves ; 6, the opening of the tricuspid valves (compare with Fig. 43).

The fall (y) in Figs. 43 and 44 is due to the blood, that has been stored in the auricle during ventricular systole, flowing into the ventricle after the opening of the tricuspid valves. Then as the ventricle becomes filled, stasis in the auricle and veins takes place, causing the rise between y and a, Figs. 43 and 44, till the auricle again contracts.

§ 109. **Standards for interpreting a jugular pulse.**—Such are briefly the main factors concerned in the production of the auricular form of the venous pulse. It is at times difficult to interpret the tracings, so that it is necessary to have definite standards to help in deciphering certain obscure features.

In Fig. 46 I have placed below the jugular pulse, tracings representing the apex beat and the carotid and radial pulses, to show the relation in time of certain events in these various movements. The numbered perpendicular

lines indicate the simultaneous events in the jugular pulse, the apex beat, the carotid, and the radial pulse. The perpendicular lines facilitate the comparison of the tracings at definite points in the cycle, and have the same significance in the later tracings: 1, the beginning of the auricular systole; 2, the beginning of the ventricular systole; 3, the opening of the semilunar valves and the appearance of the carotid pulse; 4, the beginning of the radial pulse; 5, the closing of the semilunar valves; and 6, the opening of the tricuspid valves. The time is recorded in fifths of seconds in this and other tracings.

Although the carotid pulse and the apex beat can sometimes be usefully employed as a standard, it will be found that the radial pulse is, on the whole,

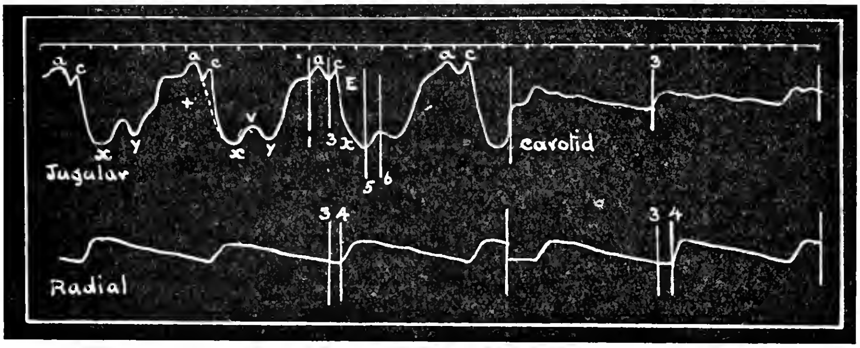

Fig. 47. Simultaneous tracings of the jugular and the radial pulses in the first part of the tracing and of the carotid and the radial in the latter part. The jugular pulse is of the auricular type. *a*, auricular wave; *c*, the carotid wave; *v*, the ventricular wave; *x*, the auricular depression; *y*, the ventricular depression. These letters have the same significance in all the other tracings, and the numbered perpendicular lines have the same significance as those in Fig. 46.

the most convenient in practice. A certain loss of time takes place in the transmission of the wave to the radial pulse, and this can be estimated by taking simultaneously with the radial a few beats of the carotid, as in Figs. 44 and 47, in which the space between 3 and 4 shows the loss of time between the appearance of the carotid and the radial pulse. This loss being allowed for, one can always find a definite period in a jugular tracing which corresponds to any event occurring in the neck due to the ventricular systole. I also employ a very useful period, namely, that portion of the ventricular systole during which the semilunar valves are open (period marked *E* in all the tracings). Its duration can be found in the radial tracing, extending from the beginning of the upstroke to near the bottom of the dicrotic notch. It corresponds to the time between the perpendiculars 3 and 5 in all the carotid, jugular, and apex tracings given here. In the neck the period *E* begins

with the carotid pulse. There is a slight delay between the opening of the aortic valves and the carotid pulse, but it is so short (one-fiftieth of a second) that it may be ignored. The space *E* in the radial tracing begins about one-tenth of a second behind the same period in the neck.

Another important standard is that of the opening of the tricuspid valves (perpendicular 6 in all the tracings), which in jugular tracings is always indicated by the beginning of the fall of the wave *v*. In the apex tracing this event occurs at the bottom of the fall, after the systolic plateau, as in Fig. 25.

§ 110. **The carotid wave.**—In the tracing in Figs. 44 and 47, in addition to the waves *a* and *v*, which have already been described, there is another wave marked *c*. In Fig. 42 it will be seen that the subclavian and carotid arteries lie in such close proximity to the jugular vein that the receiver

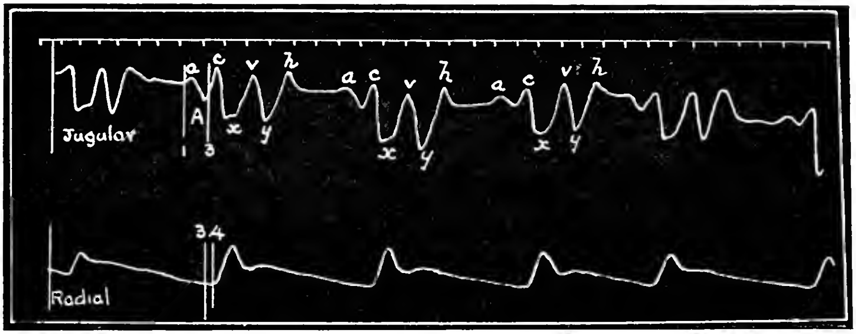

FIG. 48. Shows a diastolic wave *h* in the jugular tracing. Rate 48.

covers a portion of these arteries. In consequence of this, the stroke of the arterial pulse affects the tracing from the jugular, and produces the wave *c*, which I have called the carotid wave. A considerable amount of discussion has taken place in regard to the cause of *c*, but if an observer will carefully take tracings higher and higher up in the neck, he can easily satisfy himself of its nature, for it gradually assumes the character of a tracing from the carotid artery. The recognition of *c* as due to the carotid (or carotid and subclavian) helps much in the analysis of tracings from the neck, particularly when there is a delay between the auricular and ventricular systole, the duration of the *a-c* interval being the best measure of the delay. (Experimental observations show that faint waves may occur in the veins about the time of the carotid wave, produced in some obscure way by the systole of the ventricle, but the carotid and subclavian impact is the main, and for practical purposes the only one, that need be considered.)

The true venous curve would follow the dotted line in Figs. 46 and 47.

§ 111. **The notch on the ventricular wave.**—In a great many cases the wave *v* has a notch on it just before its termination (perpendicular line 5 in Fig. 44). It corresponds in time to the closure of the semilunar valves, and the following rise in *v* occurs between the closure of the semilunar valves and the opening of the tricuspid valves (postsphygmic interval,

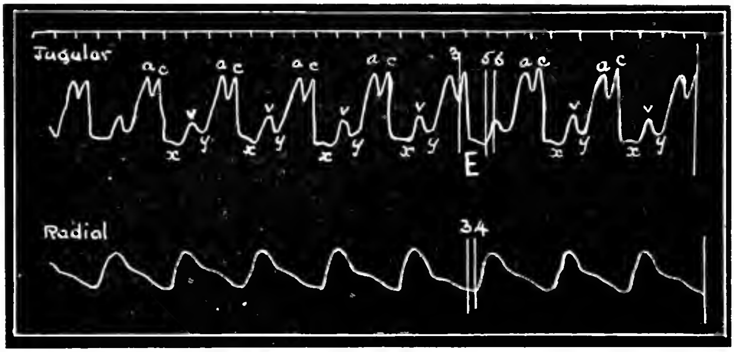

Fig. 49. In increase of the heart's rate the period between *y* and *a* becomes shortened, so that the auricular wave, *a*, follows immediately after *v* (compare with Figs. 47 and 48).

F, Fig. 43). The exact cause of this notch is still a matter of dispute—no satisfactory explanation being yet forthcoming. I have often employed it as a useful guide in measuring the period when the semilunar valves close, and it is represented by the perpendicular line 5 in all the tracings.

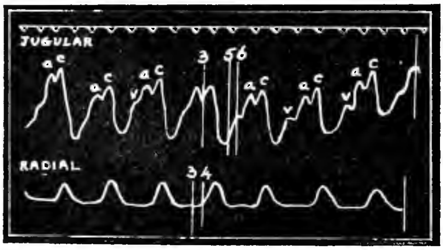

Fig. 50. With great increase in the rate the waves *v* and *a* become blended.

§ 112. **The diastolic wave.**—Occasionally a wave may be detected in slow-acting hearts shortly after the opening of the tricuspid valves (*h*, Fig. 48). A. G. Gibson [356] and Hirschfelder [362] describe this as due to the inrush of blood into the ventricle floating up the cusps and causing a transient closure of the tricuspid valves. Thayer [424] and Gibson also describe a sound heard occasionally at this time.

§ 113. **Changes due to variation in the rate of the heart.**—When the heart's rate increases the shortening of the cycle takes place mainly at the expense of the diastolic period. In the venous tracing the first effect is shown by the disappearance of the period of stasis, the wave *a* following immediately on the ventricular wave *v* (Fig. 49) ; with still greater increase in the rate, *v* and *a* become blended (Fig. 50).

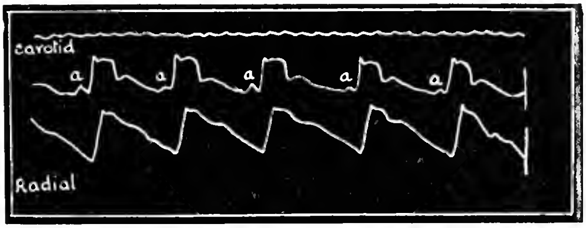

FIG. 51. In tracings from the neck the small wave, *a*, due to the auricular systole may be the only evidence of the jugular pulse.

§ 114. **Method of analysing a tracing.**—In the tracings given so far the waves have been distinct and well marked. It often happens that the jugular pulse is extremely small, so that we only get a slight movement due to the auricle, the main portion of the tracing being due to the carotid,

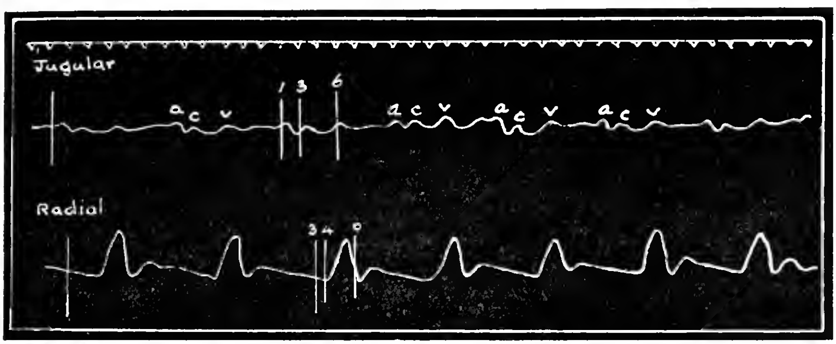

FIG. 52. The movements in the neck seemed dancing vibrations but an analysis of a tracing refers each movement to a definite cause.

as in Fig. 51. At other times the movements of the neck seem mere dancing vibrations, and the record obtainable shows a series of small undulations. But with the radial pulse as a standard, one can definitely assign each undulation to the force producing it. Thus in Fig. 52 we can analyse the tracing by the following procedure : Make a downstroke (4) parallel with the perpendicular line at the beginning of the radial tracing, at the beginning of a radial pulse beat. As the carotid pulse occurs nearly one-tenth of a second

before the radial, draw the perpendicular line 3 one-tenth of a second in front of 4. Measure the distance from the perpendicular line at the beginning of the tracing to 3. Draw a downstroke in the jugular tracing at the same distance from the one at the beginning. This will be found to fall at the beginning of a small wave, which therefore must have been due to the carotid, and so mark it *c*. The auricular wave occurs one-fifth of a second in front of *c*, and so the wave *a* can only be due to the auricular systole. In Fig. 46, p. 111, it was shown that the opening of the tricuspid valves (perpendicular line 6) often coincides with the bottom of the dicrotic notch in the radial tracing. If now a perpendicular line (6) be drawn at this

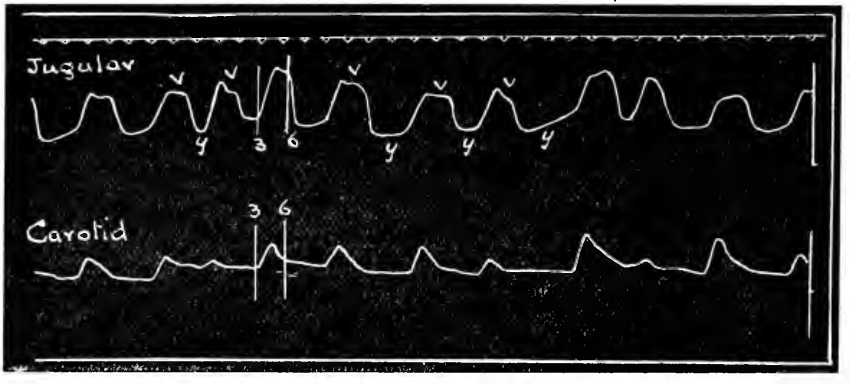

Fig. 53. Simultaneous tracings of the jugular and carotid pulses, showing one large wave *v*, synchronous with the carotid pulse, and due to the ventricular systole, and one large fall, *y*, synchronous with and due to the ventricular diastole. There is no sign of an auricular wave, and the jugular pulse is therefore of the ventricular type, and the rhythm of the heart is continuously irregular.

period in the jugular tracing, it will be found to fall at the end of a wave, *v*, which must therefore be the ventricular wave.

By strictly following such a method as the foregoing, little difficulty will be experienced in analysing the great majority of tracings.

§ 115. **The ventricular form of the venous pulse.**—In Figs. 53 and 54 are tracings of the jugular pulse. At a glance these are recognized to be totally different from the form of the venous pulse just described. The waves, *v*, in Figs. 53 and 54 are due to the blood being forced back through the tricuspid orifice into the veins by the contraction of the right ventricle. In a sense its origin is identical with the wave, *v*, in the auricular venous pulse as in Figs. 44 and 45, but appears earlier in the cardiac cycle (synchronous with the carotid pulse) because *there is not now a dilating auricle interposed between the vein and ventricle.* When we come to analyse tracings of the ventricular venous pulse with a standard movement, as in

Fig. 53, we discover there is no evidence of an auricular wave, nor of a fall corresponding to the fall x in the auricular venous pulse—in other words, there is one great wave (v) synchronous with the ventricular systole, and one great fall (y) synchronous with the ventricular diastole. Another point to be noticed is that the rhythm frequently is irregular, and that when there is a long diastolic period there is a rise in the tracing due to the vein filling, as after the long pauses in Fig. 54.

There are two conditions which produce the ventricular form of the venous pulse :—

(1) Great distension and paralysis of the right auricle. Here there is great engorgement of the right heart so that the auricle becomes embarrassed

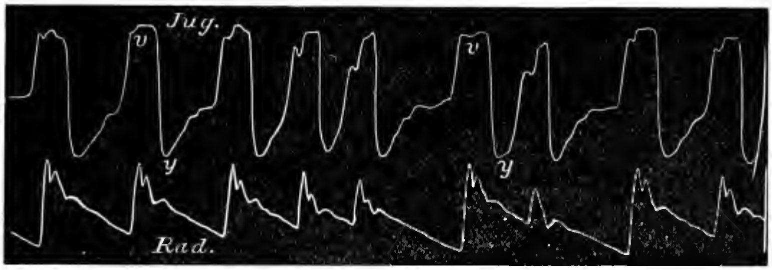

FIG. 54. Simultaneous tracings of the jugular and radial pulses, showing the ventricular jugular pulse, i.e. one large wave, v, synchronous with and due to the ventricular systole, and one large fall, y, synchronous with and due to the ventricular diastole. The rhythm of the heart is continuously irregular. When there are long pauses the tracing of the jugular gradually rises before the next large wave, on account of stasis in the veins. When the pause is short there is no sign of stasis, or only a slight wave, as on the second beat, where it might simulate a wave due to the auricle, but the real cause is seen to be stasis in the veins when the pause is longer.

in its action, and the a wave in the jugular pulse diminishes in size and disappears, while the v wave increases in size and occupies the whole period of ventricular systole. With recovery and diminution of the engorgement the auricular wave reappears. It is to be noted that the rhythm in these cases is always regular. This manner of production of the ventricular venous pulse is however very infrequent, far less common than what my earlier observations led me to suppose, for the vast majority of cases of the ventricular venous pulse are due to

(2) A change in the starting-point of the heart's contraction. In consequence of its great importance I deal with the matter in detail, and adduce further proof of the nature of the change in Appendix II. For the sake of brevity, I summarize here the facts which show *that the ventricular venous pulse is usually an evidence of the starting of the rhythm of the heart at some place other than at the mouth of the veins.* For reasons to be given later, I assume that this starting-place is in or about the a.-v. node (Fig. 2),

and hence refer to this rhythm of the heart, where there is a ventricular venous pulse, as the 'nodal rhythm'.

The onset of the nodal rhythm is characterized by :—

(*a*) Change of the venous and liver pulses from the auricular type to the ventricular.

(*b*) The disappearance of all other evidence of the auricular systole from its normal place in the cardiac cycle (as the disappearance of a presystolic murmur, and the absence of an auricular wave in the tracing from the apex beat).

(*c*) In many cases there is a continued irregularity in the heart's action.

It so happened that my earlier cases of the ventricular form of the venous and liver pulses were all secondary to disease of the mitral valve, and at the post-mortem examinations I found the right auricle enormously distended and the muscle-fibres greatly atrophied, so that I surmised that the auricle, like an over-distended bladder, had become paralysed. This view seemed to be confirmed by the results of careful observation of individual patients extending over many years, the auricular wave never putting in an appearance at its normal period in the hundreds of tracings I had taken. This view I expressed in my book on the Pulse [164], but shortly after writing it a few cases came under my observation that gave me grounds for suspecting that 'auricular paralysis' was not the only condition that gave rise to the ventricular form of the venous pulse.

I therefore began an inquiry into all forms of arrhythmia, and collected a large number of cases. From amongst these I was able to differentiate a group of over six hundred cases in which the irregularity was of a disorderly nature, as in Figs. 53 and 54. Over one-half of these cases had a venous pulse, and in every case it was of the ventricular form. In more than fifty cases the disorderly rhythm had started while the patient was under observation, and it invariably accompanied the sudden change of the jugular pulse from the auricular form to the ventricular.

In a careful analysis of a great number of my cases, I found that in some there was only the large wave, lasting from the time of the opening of the aortic valves to the opening of the tricuspid valves, as in Figs. 53 and 54. On the other hand, there were others in whom there could be seen in the jugular veins in the neck two movements during ventricular systole—the first movement short and abrupt, followed by a larger movement. These two movements appeared in the tracings (a' and v in Figs. 55, 56, 210, and 211). The time of the appearance of a' was exactly that of the carotid in the majority of cases (perpendicular line 3 in Fig. 55). In a few instances I found this wave a little earlier. Thus, in Fig. 56 it appears before the

carotid and corresponds with the beginning of ventricular systole, as is shown in Fig. 57. The patient from whom this tracing was taken is described in the Appendix II (Case 7), and she had shown this form of jugular pulse

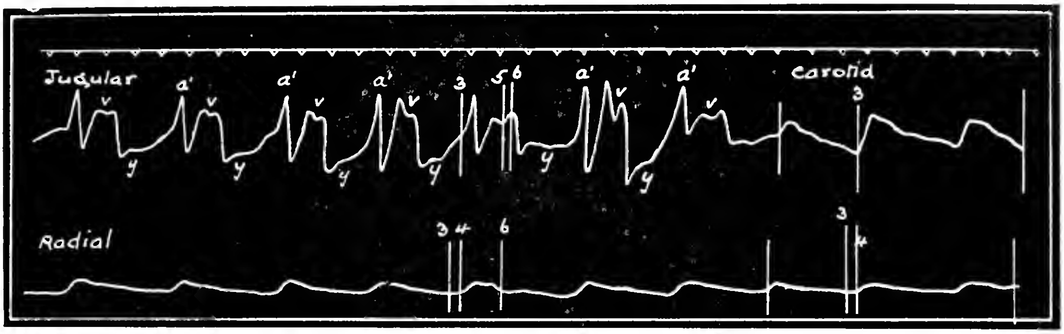

Fig. 55. The jugular pulse is of the ventricular form and shows two waves, a' and v, during the period of ventricular systole (3 to 6). The wave a' appears at the same time as the carotid pulse (perpendicular line 3), and is due to the auricle contracting during the ventricular systole. There is a notch in the v wave (perpendicular line 5) corresponding to the notch in the v wave in Figs. 44 and 45.

for over five years. At the post-mortem examination, the taenia terminalis of the auricle was hypertrophied; it can therefore be inferred that the auricle must have contracted. There was never any sign of the auricular

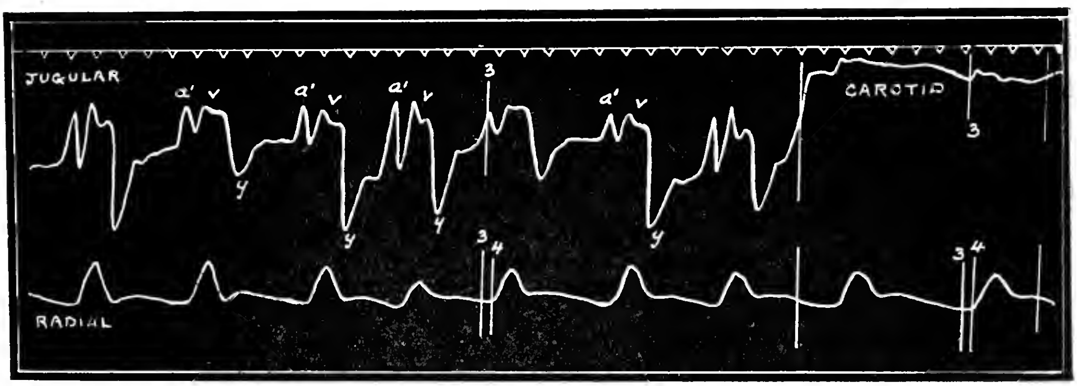

Fig. 56. Ventricular venous pulse. There are two waves (a' and v) in the jugular pulse, and the wave a' appears slightly before the carotid (perpendicular line 3), and is due to the auricle beginning to contract at the same time as the ventricle.

wave before the carotid pulse, and I therefore concluded that the wave a' in Figs. 56 and 57 must have been due to the auricular systole, and that in these cases the auricle and ventricle contracted almost simultaneously (see also Case 12, Appendix II).

Assuming this interpretation to be correct, it will be found that the wave *a′* appears at the same time as the carotid (perpendicular line 3 in Fig. 55). As the ventricle is in systole before the aortic valves open, the ventricular contraction must have preceded the contraction of the auricle which produced the wave *a′*. On the other hand, in such a rare instance as that shown in Figs. 56 and 57 the auricle and ventricle must have started together.

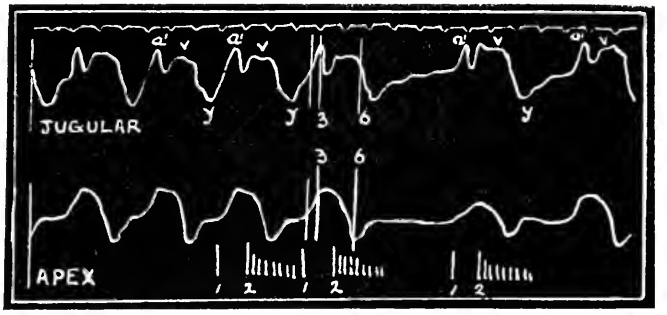

FIG. 57. Tracings of the ventricular venous pulse and of the apex beat. There are two waves, *a′* and *v*, in the venous pulse, and the wave *a′* is seen to appear at the beginning of ventricular systole. The sounds of the heart (1 and 2) are represented under the apex tracing, and the shading after 2 represents the mitral diastolic murmur. During the short pause it fills up the whole period between the first and second sounds, whereas when the pause is long, it stops some distance before the first sound.

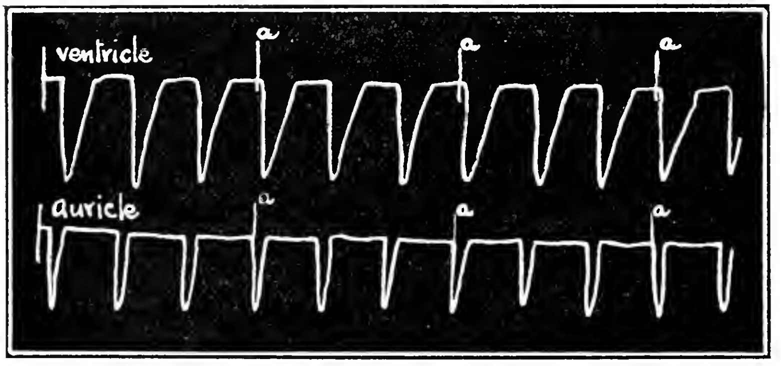

FIG. 58. Tracings showing the normal ventricular and auricular movements from a dog's heart. The downward movement represents the contraction. The perpendicular lines, *a*, indicate the beginning of auricular systole, and, in the ventricular tracing, are seen to *precede* the ventricular contraction. Compare with Fig. 59. (Cushny.)

A remarkable confirmation of this view has been recently obtained by Cushny [187] and Lewis. In experimenting on the dog they have been able to produce in various ways an abnormal rhythm of the heart agreeing

exactly with the description given above—that is, the ventricle started one-tenth of a second or less before the auricle (see Figs. 58 and 59), or the ventricle and auricle started simultaneously. Lewis has demonstrated the disappearance of the auricular systole from the normal place in the cardiac cycle in cases of nodal rhythm by means of electro-cardiograms (see Fig. 279, Appendix VII).

§ 116. **Conditions giving rise to a venous pulse.**—To a great extent we are even at this day quite at a loss to explain all the conditions that give rise to a venous pulse. Most people in good health show it, while in cases of marked heart failure it may be entirely absent. Some people when in

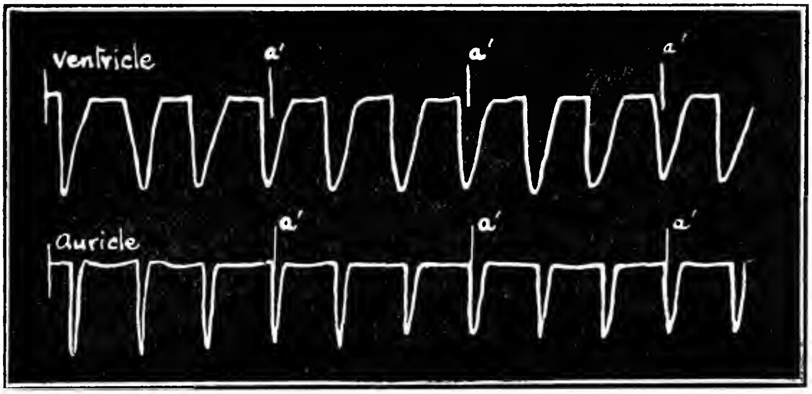

FIG. 59. Tracings of the movements of the ventricle and auricle from a dog's heart, poisoned by aconitin. The perpendicular lines, a', indicate the beginning of auricular systole, and in the ventricular tracing are seen to *follow* the beginning of the ventricular contraction. Compare with Fig. 58. (Cushny.)

robust health show no signs of it, but if they become slightly debilitated the venous pulse may appear in the neck. In some cases of pernicious anaemia the venous pulse may be a very prominent symptom, in others it may never appear. Some women during pregnancy develop a large venous pulse, others only develop it during the puerperium, while others never show the slightest sign of it. In irregular action of the heart it may assume in certain cases, enormous proportions ; in other cases there is not the slightest sign. A man may show it during one attack of heart failure, and during another and more grievous attack it may be absent.

I have endeavoured to find out the reason for this variability, but though in some cases I have been able to connect its appearance and disappearance with definite changes in the heart, yet on the whole the matter is one which still puzzles me.

CHAPTER XIV

Enlargement and Pulsation of the Liver

The symptoms arising from enlargement of the liver due to heart failure receive little consideration from clinicians, and are very frequently overlooked or misunderstood. To a certain extent, this is due to the fact that this enlargement may appear at such an advanced stage of cardiac failure that the diagnosis and treatment can be determined without paying particular attention to the liver symptoms. Graham Steell [180] includes the enlargement of the liver as one of the cardinal symptoms of heart failure, and Salaman [428] has given a very suggestive analysis of the pathological changes and the conditions inducing them ; but clinicians generally have dealt with the subject in a most perfunctory manner. While it is true that the conditions producing these liver troubles imply an advanced stage of heart failure, yet the recognition of the symptoms have an important bearing on diagnosis and treatment in many cases. The symptoms due to changes in this organ are not always easy to understand, but that is no reason for ignoring them.

§ 117. **Reflex or protective symptoms.**—Usually in the early stages of liver enlargement we find evidences of the intervention of the protective mechanism (see Chapter VI). While the liver may be only one or two inches below the ribs, the muscle wall of the upper part of the right half of the abdomen becomes hard and tender. This tenderness is invariably put down to the liver itself, and the manner in which this 'tenderness' is usually demonstrated is by eliciting pain on pressing the finger into the patient's abdomen. But if the extent of the hyperalgesia and the size of the liver be mapped out, it will be found that the former is far more extensive than the latter, and sometimes extends round to and affects the

erector-spinae muscles. In some cases the skin and subcutaneous tissue also become tender, but they rarely become so sensitive as the muscles. Sometimes there seems an increased tenderness where the liver is reached, but this will be found to be due to the more effective compression of the muscle between the finger and the liver. There are many other ways by which the resourceful observer can demonstrate the tissue in which the tenderness is present.

The consequence of this muscular hyperalgesia is manifested in various ways. If the patient is going about he may suffer severe pain across the upper part of the abdomen or in the back, this probably being due either to increased engorgement of the liver or to the increase of the pain in consequence of the exercise of the hyperalgesic muscles. This tenderness and rigidity of the abdominal muscles interferes with the respiration of the patient. He cannot breathe deeply, and attempts to do so are painful, hence there results rapid and shallow breathing, with further embarrassment of the right heart and a tendency to pulmonary stasis.

With long-continued persistence of the enlargement, all the sensory phenomena disappear, the abdominal wall becomes lax, and sometimes the edge of the liver can be grasped.

§ 118. **Signs of enlargement of the liver.**—It is not always easy to make out the enlargement of this organ. The contracted muscles often prevent the palpation, and even percussion helps but little. Ascitic and gaseous distension add further to the difficulty. But with care and gentleness in palpation one may overcome the resisting muscles. Even when the edge of the liver cannot be made out, the peculiar sense of resistance conveyed to the exploring hand may reveal the enlarged liver. Other methods may be adopted, such as pushing the liver forward with one hand while the other explores the front. When the muscles relax and the hyperalgesia disappears, there is no difficulty in finding out the enlarged liver, except when there is great distension of the abdomen.

§ 119. **Pulsation of the liver.**—When the liver is enlarged from heart failure it not infrequently pulsates. If the abdominal muscles are lax, there is no difficulty in recognizing this. If one hand presses on the liver behind and the other is laid on it in front, the latter is heaved up and down with the pulse. Even where there is a considerable amount of muscular contraction, exploring the edge of the liver with the liver receiver will often reveal the pulsation (§ 71).

There are two forms of liver pulse, corresponding to the two forms of venous pulse—an auricular, Fig. 60, and a ventricular, Fig. 61, the latter being an evidence of the nodal rhythm. When a liver pulse and a jugular pulse

are both present in the same individual, they are always of the same form. In Fig. 62 a tracing of an auricular jugular pulse is taken simultaneously with a tracing of the auricular liver pulse. In Fig. 63, the tracing of the

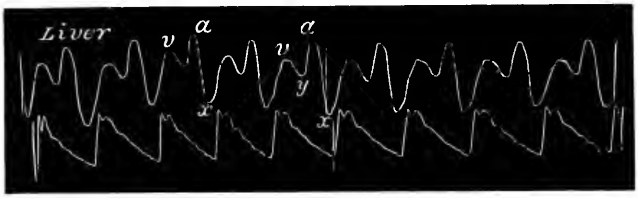

Fig. 60. The liver pulse is of the auricular form and shows a well-marked auricular wave, *a*.

ventricular jugular pulse is seen to be identical with the liver pulse in Fig. 61, both tracings being from the same patient.

§ 120. **Conditions producing enlargement and pulsation of the liver.**—I have already remarked on the variety of conditions producing the jugular

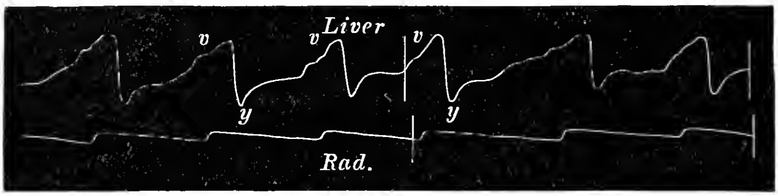

Fig. 61. The liver pulse is of the ventricular form, showing no auricular wave.

pulse, and there is a like difficulty in understanding the conditions which give rise to liver enlargement. Cases otherwise identical in their symptoms, and suffering from heart failure due to the same cause, may differ in this

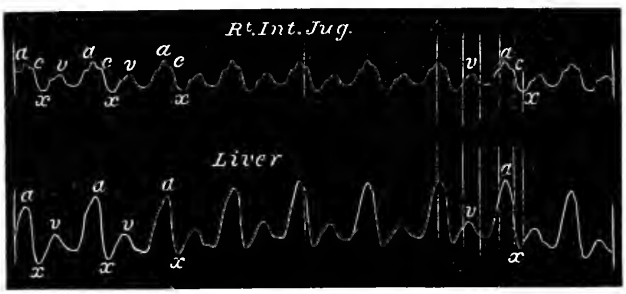

Fig. 62. Simultaneous tracings of the jugular and liver pulses, showing the correspondence between the waves *a* and *v*, and the absence of the carotid wave *c* from the liver pulse.

particular, some showing enlargement of the liver and others failing to do so. Similarly it is not always quite clear why some pulsate while others do not. To a certain extent, I think this is due to the condition of the right

auricle. It takes some force to distend the liver, and normally the right auricle has not sufficient strength, so that as long as the right auricle contracts and dilates in its normal place in the cardiac cycle it prevents the ventricle exercising its force on the liver. When, however, the ventricle starts the rhythm, it drives the blood through the incompetent tricuspid orifice with such force that the liver pulsates. It is in the cases of nodal rhythm that we most frequently find the liver pulsating. When the nodal rhythm is transient the liver may quickly enlarge and pulsate, and with cessation of the attack as quickly subside and cease to beat—the liver pulse being of the ventricular form (Fig. 207).

Reasoning that it requires some force greater than the normal strength of the right auricle to produce pulsation of the liver, I at first drew the

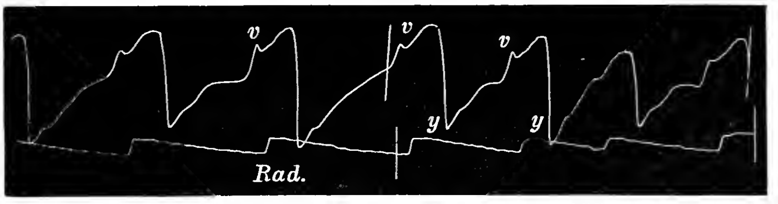

FIG. 63. Simultaneous tracings of the jugular and radial pulses. The jugular pulse is of the ventricular form and is identical with the liver pulse in Fig. 61, both being taken from the same patient.

conclusion that an auricular liver pulse indicates hypertrophy of the right auricle, and as this occurs most characteristically in cases of tricuspid stenosis, I regarded the auricular liver pulse as diagnostic of tricuspid stenosis. All the cases that had shown this auricular liver pulse during life, whose hearts I examined *post mortem*, showed tricuspid stenosis. But I have now had a number of cases showing this form of liver pulse, in which I doubt if I am justified in assuming tricuspid stenosis. Volhard [372] describes the auricular liver pulse in pericardial effusion, and Wenckebach [437] in a case of adherent pericardium.

§ 121. **Jaundice.**—Jaundice is a frequent accompaniment of enlargement of the liver; though of itself of little importance, it should be kept in mind that a slight jaundice may be misleading. Many patients with advanced heart failure and with the nodal rhythm get rapidly thinner. In these the liver is sometimes greatly enlarged, so that the wasting of the patient, the enlargement of the liver, and the jaundiced tint, present the features of malignant disease of the liver, and I have seen cases presenting these symptoms thus wrongly diagnosed.

§ **122. Differential diagnosis.**—Many writers refer to an 'arterial pulsation' of the liver, and I have often wondered what they mean, particularly as no details are ever given. I have never found any condition that could come under this heading, and I suspect that some observers have mistaken the movement of the liver, when it is pulled up and down with the systole and diastole of the ventricle, for a pulsation, particularly as it is sometimes mentioned in connexion with aortic regurgitation. Wherever there is much cardiac enlargement, movement of the liver is produced. Even in patients with normal hearts but with lax abdominal wall, this up-and-down movement can be recognized. It has already been referred to in § 81. Here I need only point out that the conditions in which it occurs are generally very different from that of the heart failure in which the true liver pulsation occurs. A tracing of this movement

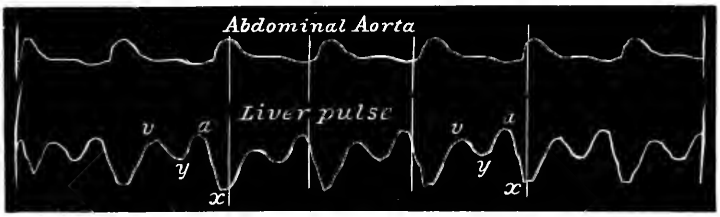

Fig. 64. Tracings from the abdominal aorta and the liver. The liver pulse is of the auricular type.

sets the matter at rest, as the fall due to the ventricular systole does not begin until the opening of the aortic valves and the expulsion of blood from the chest, which is practically synchronous with the carotid pulse. In the ventricular form of liver pulse, on the other hand, there is a rise during ventricular systole. A tracing of the liver movement can be distinguished from the auricular liver pulse in that the fall in the latter case precedes the carotid pulse.

There is generally little difficulty in distinguishing the pulsation of the abdominal aorta from that of the liver pulse. In cases where the ventricular liver pulse is present, it is very rarely that the abdominal aorta can be felt, and the characters of the tracings are not likely to be confused. The auricular form of liver pulse is still more distinct in character from that of the abdominal aorta, as shown in Fig. 64.

§ **123. Prognosis.**—Liver enlargement and pulsation from cardiac disease indicate a very advanced stage of heart failure. In cases where the heart failure is secondary to a rheumatic affection of the heart with mitral

disease, it may only appear during the attacks of heart failure to which the patients are liable. The enlargement subsides with improvement, and if the heart's restoration be good the patient may have no signs of the liver enlargement for many years. Some patients with the nodal rhythm

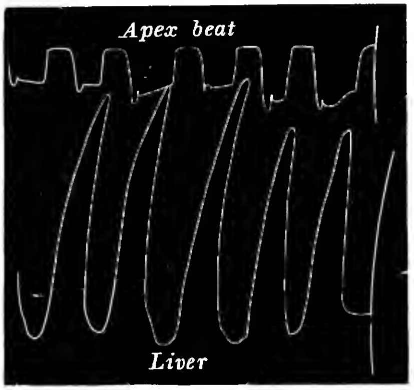

FIG. 65. Simultaneous tracings of the apex beat and of the liver pulse. The liver edge was below the level of the umbilicus, and the pulsation of great size.

secondary to rheumatic affection of the heart may for years have a big pulsating liver (Fig. 65), in others the enlargement appears during temporary heart failure. In some of these the exhibition of digitalis may cause the subsidence of the liver in a couple of days. Fig. 66 was taken from the liver

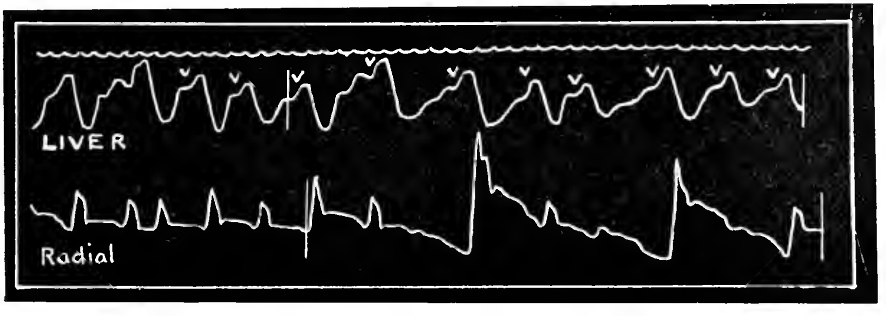

FIG. 66. Tracings of the liver and radial pulses during a period of extreme heart failure (nodal rhythm).

of a man which was pulsating as low as his umbilicus. In a couple of days all signs of the liver below the ribs had gone. The enlarged liver coming on in paroxysmal tachycardia, i.e. transient nodal rhythm, is a very serious symptom, as it indicates marked dilatation of the heart. Also in the heart failure due to cardio-sclerosis with the permanent nodal rhythm, it is a very

grave symptom, as the heart failure is due to extensive degeneration of the cardiac muscle, and therefore does not respond to the influence of rest and digitalis. Enlargement of the liver in muscle failure due to chronic alcoholism is a bad sign.

§ 124. **Treatment.**—Though no treatment is directed specially to the liver enlargement, the effect of the rigid tender muscles in embarrassing the respiration should be borne in mind, and suitable attempts made to put the patient in a comfortable position so that he can breathe more easily, and the pain due to the enlarged liver should call for the cessation of all exertion that induces it. A smart mercurial purge may sometimes afford considerable relief.

CHAPTER XV

Increased Frequency of the Heart's Action

§ 125. **The normal rate.**—The rate of the heart varies very considerably according to age, sex, and individual peculiarities. At birth, the rate is usually from 130 to 140 per minute. With advancing years the heart slows down gradually: between nine and ten the average is about ninety; at twenty, about seventy-four; at thirty, from sixty-six to seventy-six: it remains at about this latter rate until over fifty, when it gradually begins to increase in frequency. At all ages very considerable variations may be met with.

The heart's rate is greatly increased by exertion, even in those who are in good training. Deane found the pulse-rate over 200 per minute in a professional dancer, at the end of a dance. It quickly subsided to the normal rate. According to Pembrey and Todd the increase in rate is somewhat greater in the trained than in the untrained, but the decrease in the rate to normal is more rapid in the trained man.

§ 126. **Classification.**—In considering the conditions that give rise to increased frequency of the heart's action, we are confronted with such a great number that it is impossible to deal with them all. What I propose to deal with here is the abnormal increase of pulse-rate, and certain conditions other than febrile which induce the rapidity of the pulse. These may fittingly be discussed in three groups: (1) Those cases in which the heart responds to a call upon its energy by increased frequency; (2) those cases in which the heart-rate is continuously increased; (3) those cases in which the periods of increased rapidity take place in irregular paroxysmal attacks.

§ 127. **Cases which respond to a call upon the heart's energy by increased frequency.**—The cases in the first of these groups, those in

which the heart responds to an increased call upon its energy by increased frequency, show in reality but an exaggerated form of the normal condition. When we find that a patient is seized with palpitation or rapid heart action after mounting a few steps, we recognize as abnormal that which would have been regarded as normal in an individual who had run half a mile at the top of his speed. In other words, this increased rate is an evidence that the field of the heart's response to effort is greatly reduced. A further deduction can be made from observing these patients, viz. that exhaustion of the reserve force heightens the excitability of the whole heart, for not only is the rate increased, but the contraction sweeps through the heart with greater rapidity and the systoles of the chambers are of shorter duration. The increased frequency should always lead one to seek the cause from which it arises. The conditions underlying it are too numerous to mention, but they all in the end point to enfeeblement of the muscle of the heart. In all exhausting diseases, and after convalescence from such a wasting sickness as typhoid fever, the heart's rate can be greatly increased by even very moderate exertion. In the various anaemias (chlorosis, pernicious anaemia, malignant cachexia) the rapid heart action is very often the symptom to which the patient's attention is first called. In organic affections of the heart, as in the various forms of myocarditis, in fatty degeneration of the myocardium, and in valvular disease when there is only a small amount of reserve force in the muscle, increased frequency of the pulse on exertion is extremely common. Many of the patients whose ailments are included in the foregoing groups, when at rest, have a pulse beating about or not much above the normal rate. The heart then seems to be capable of sustaining the demands of the circulation, but seems to be working near the top of its reserve energy. On exertion this reserve energy is speedily exhausted, and in order to make up for its inability to respond to the demand of the tissues for more blood by giving stronger ventricular contractions, it responds by giving a greater number of feebler and less complete contractions.

In addition to the increased rate there is usually hurried and laboured respiration, and this too occurs whether a strong heart is overstrained by a great effort, or a weak heart by a slight effort. Not infrequently in elderly people, before the interference with respiration can arise, the patient in making an effort is stopped by a feeling of weight or oppression within the chest, or even by pain striking across the chest, sometimes severe, sometimes slight, but in all cases imperiously demanding a cessation of the effort.

It is as impossible to indicate with any approach to accuracy when a pulse-rate is abnormal on moderate exertion as it is to indicate what the pulse-rate should be in health under similar circumstances. The increase is often

so marked that its recognition is beyond dispute. Thus, in making a patient sit up or turn over in bed, a rise of five to ten beats a minute may not be worthy of much attention, but if the increase is fifteen to thirty beats then there is distinct evidence that we have to do with some condition that has exhausted the heart's reserve power. This increase of the pulse-rate beyond the normal on moderate exertion does not give any clue to the nature of the condition that has reduced the heart's reserve power. As already indicated, these conditions are so numerous that an examination for other symptoms must be undertaken to discover them.

§ 128. Cases in which the heart's rate is continuously increased. (*a*) *Valvular disease.*—The second division, in which the heart maintains a frequency beyond what we recognize as within the limits of health, also includes a great variety of heart conditions. We have among these the series of valvular diseases of the heart, with the muscle exhausted, it may be from struggling against the obstruction caused by the valve lesion, or with the muscle itself degenerated. Not only do such hearts respond to effort with marked increase in frequency, but even during rest the heart may beat with abnormal rapidity, regularly or irregularly. This forms a very important factor in arriving at an estimate of the strength and condition of the organ. The other symptoms of heart failure present will help in indicating the stage at which the patient has arrived.

(*b*) *Affections of the myocardium.*—Apart from patients with manifest valvular disease of the heart, there are many whose pulse is rapid, and in whom no disease of the heart can be detected by physical signs. The chest-wall may be thick and fat, or the lungs so voluminous that the actual size of the organ cannot be satisfactorily defined. The sounds, though free from murmur, may be so slightly modified that no certain inferences can be drawn from them. Yet that serious mischief is present is but too often demonstrated by watching the after-history of these cases. If we exclude for the present the consideration of certain neurotic conditions, the cause of the quickening in all cases is really associated with a want of strength in the muscular wall. In valvular disease this is usually spoken of as failure of compensation. In degeneration of the wall, fatty or fibrous, the weakening of the wall is directly due to this degeneration. In the great series of hearts overstrained from excessive exertion, the weakness of the wall is the principal cause of the whole train of symptoms associated with the failure of the heart. In arriving, therefore, at an estimate of the value of the pulse quickening, a consideration of the other symptoms present will be necessary to recognize what is the cause of the increased pulse-rate in each special case. The circumstances, age, and condition of the patient,

will help much in recognizing the rapid pulse due to actual degeneration of the heart-wall. But there is a series of cases in which it is difficult to account for the rapid pulse, especially when it occurs in the apparently strong in the prime of life. In these cases there is generally a history of hard work or periods of excessive muscular exertion. Sometimes the condition receives a special name, as ‘ the soldier’s heart ’. Medical men whose practice lies amongst workpeople subjected to such muscular exertion are familiar with a similar condition. The heart overstrain is most evident amongst those with a tendency to obesity and who indulge rather freely in alcohol. The symptoms are mainly a quickened pulse and shortness of breath on exertion. Examination of the chest is often fruitless, the chest being large and deep, and the lungs often voluminous. Much improvement results from care and rest, and appropriate treatment.

(*c*) *Pregnancy.*—It may be noted that these symptoms are often present in pregnant women, but there the cause is but temporary, and a certain amount of recovery follows delivery. But in my experience there is often left a certain amount of cardiac weakness, shown by a distinct limitation of the field of cardiac response.

(*d*) *Alcohol.*—In all obscure cases of rapid heart action, the question of over-indulgence in alcohol should be carefully inquired into. The patients often try to hide their habits in this respect, but the physician can generally find a clue in the manner of the individual, his facial aspect, tremulous muscles (especially the tongue), want of appetite or nausea in the morning, and that *tout-ensemble* which leads the experienced physician to suspect the secret alcoholic. Accompanying the rapid pulse and other phenomena there are often a sinking sensation in the epigastrium and sense of exhaustion on exertion, and breathlessness. The heart may be only slightly enlarged, or there may be great dilatation, usually accompanied by enlargement of the liver and tenderness of the tissues covering it. With abstinence from alcohol these cases in the early stages quickly recover, but with continuance of the habit all the characters of severe heart failure supervene.

(*e*) *Neurotic cases.*—There is a group of people who exhibit a rapid pulse in whom no heart lesion can be detected, and whose future history demonstrates that no serious cardiac lesion existed. These people exhibit other symptoms more prominently associated with the nervous system, and are described in Chapter VIII.

(*f*) *Exhausting diseases.*—It is always well to bear in mind that a persistent quick pulse may be the earliest symptom of an attack of tuberculosis or of the onset of malignant disease. A patient may complain for months of weakness, with an absence of the sense of well-being, with no actual

suffering. After a time the development of other symptoms demonstrates the cause of the abnormal pulse-rate. In the young, particularly, persistent high frequency of the heart's action in the absence of any demonstrable heart lesion should always awaken the suspicion of a latent tubercular affection. I have watched such cases for months, and puzzled over the cause of the rapid heart-action, until the true nature of the complaint was revealed by some definite sign such as the pointing of a psoas abscess or a pulmonary haemorrhage. In most exhausting diseases (malignant diseases, pernicious anaemia, tuberculosis) the pulse-rate is continuously quickened. The frequent heart-action may usually be taken to indicate a severe infection and a serious condition.

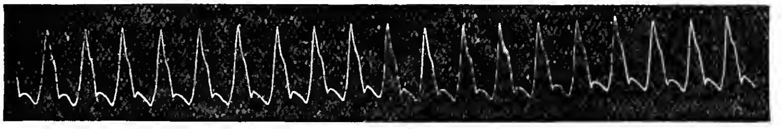

FIG. 67. From a female aged forty, suffering from exophthalmic goitre. Rate 120.

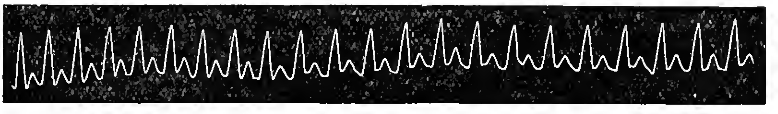

FIG. 68. From a female aged twenty-two, suffering from exophthalmic goitre. Rate 120.

(*g*) *Exophthalmic goitre.*—The essential features arising from the circulation in many cases of exophthalmic goitre, it seems to me, are the abnormal and persistent dilatation of the arterioles, and a heart acting with a force relatively great to the resistance opposed. These are indicated by the rapid and forcible pulse-wave felt by the finger, and the visible pulsation of the superficial arteries and the carotid. The corresponding sphygmographic features are a high upstroke and rapid fall, so that the dicrotic notch is near the base line (Figs. 67 and 68). The rate of the pulse may be greatly increased, up to 140–160 per minute. The same factors, the unusually forcible injection of the blood into the arteries of low blood-pressure, are present in aortic regurgitation. Though the beating of the carotid is due to similar causes in the two cases, the low arterial pressure at the end of diastole is different. In exophthalmic goitre the dilatation of the arterioles and capillaries is the sole cause, whereas in aortic regurgitation there is in addition the backward flow into the ventricle through the incompetent valves. The condition of the circulation in exophthalmic goitre is also comparable to that in some forms of sthenic fever, where the heart beats forcibly and the arteries are relaxed.

Another evidence of the relaxation of the arterioles is to be found in the

subjective sensation of warmth felt by some sufferers from exophthalmic goitre. They rarely complain of cold in winter, however lightly clad they are, and this is not infrequently the cause of matrimonial disputes, for while the ailing wife feels warm in bed during winter with few blankets, the healthy husband feels the cold keenly. This feeling of warmth has supplied me with the indications for the only treatment of this class of case that I have found both grateful and beneficial to the patient, namely, the periodic stimulation of the vaso-motor nerves by cold baths. Whenever the feeling of warmth has been present I have found these baths do good, and when there is nervousness and muscular tremor the administration of the bromide of ammonium has been of great service.

§ 129. **Cases in which the increased frequency of the heart occurs in irregular paroxysmal attacks.**—This class includes cases of 'palpitation' and 'paroxysmal tachycardia'. There are quite a number of different conditions included under these terms, and no clear idea is usually given of what is meant. A very useful and practical division may be based on the manner in which the heart's contraction starts. In the vast majority of cases of transient rapid heart-action, the heart's action is perfectly normal; the rapid action of this class will be spoken of here as 'attacks of palpitation'. In another class of patient the heart's contraction does not start at the normal place; to this latter class the term 'paroxysmal tachycardia' is limited, and the cases are described in the sections on 'nodal rhythm' (p. 309) and auricular tachycardia (p. 334).

The rate in the first class rarely exceeds 170 beats per minute, and the rhythm is regular except for the presence of an occasional extra-systole; in the latter class the rate may at first exceed 200 beats per minute, and the rhythm is frequently irregular. (Concerning the records of extreme frequency sometimes quoted, I may say I have never met with cases approaching 300 per minute, nor have I come across a single instance of a published tracing recording such a speed. They may be published, but I have not seen them. I have a suspicion of any estimate made of a pulse-rate over 200 unless graphically recorded, because I doubt if the human mind is capable of accurately distinguishing between events occurring at a speed over 200 per minute, and I doubt if any one could articulate or mentally distinguish the individual numbers at a rate of 300 per minute.)

In the following paragraph I describe under 'palpitation' the more common forms of temporary rapid action of the heart.

Palpitation.—This may occur in people suffering from a great variety of complaints. The patient is usually conscious of the change in the heart's action, feeling the rapid beats and sometimes describing them as gentle,

sometimes as hard and hammering. These latter sensations may occur with little or no increase in frequency. In cases of valvular disease with limited reserve force, slight physical effort or mental excitement may readily induce an attack. Even in the healthy, certain mental states may induce an attack, while when the system is weakened from disease the liability to attack is much increased. It is in certain neurotic subjects, particularly females, that one sees the complaint attain its most distinctive features. There may be no organic affection of the heart, and though frequent attacks may ultimately induce exhaustion of the reserve force,

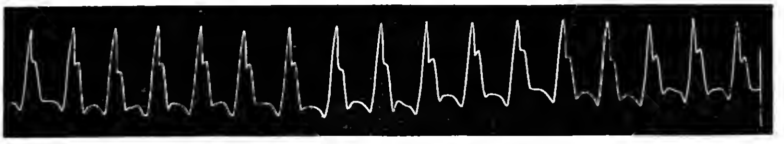

FIG. 69. During an attack of palpitation. Rate 105.

yet, as a rule, they do not appreciably shorten life. Anything that startles the patient, whether a sudden noise or mental perturbation when awake, or uncomfortable dreams when asleep, readily induces an attack. But it may supervene from more obscure causes, evidently caused by reflexes from organs more or less remote (stomach, uterus), or from undiscernible sources. When a severe attack comes on the patient may become painfully aware of the violent action of the heart. She prefers to sit upright, draws deep inspirations, and moves uneasily from side to side, with the hand pressed over the heart. It is accompanied by sensations of a distressing nature, such as a sense of suffocation, and a fear of impending dissolution. When it subsides it leaves the patient exhausted.

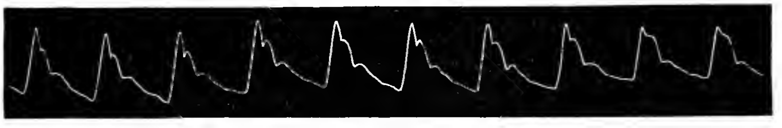

FIG. 70. Tracing of the normal pulse of the patient from which Fig. 69 was obtained. Rate 64.

During the attack the pulse is usually increased in frequency. The artery may be of fair size ; sometimes, however, it is very small. The impact of the pulse-wave on the finger is sudden and sharp and of extremely brief duration. The tracing Fig. 69, taken during an attack of palpitation, shows a high upstroke with a great fall, so that the arterial pressure at the bottom of the dicrotic notch is nearly as low as at the end of the diastolic period—an evidence that in addition to the excited heart there is great relaxation of the arterial wall. Fig. 70 is from the same patient when the heart was acting quietly.

We occasionally meet with patients in whom the pulse is extremely rapid for a period, sometimes for a few minutes, sometimes for a few hours, with no other sensation than that of exhaustion, the attack quietly subsiding (Fig. 71). The causes are so obscure that it would be mere guesswork, in the majority of cases, to attribute it to any one cause.

§ 130. **The cause of increased frequency of the heart's action.**—Apart from cases due to nerve excitation, and the nodal rhythm, it is extremely difficult to account properly for this abnormal quickening. All the parts of the heart participate in the excitability. It is not due merely to a dilatation of the heart, for we may have hearts greatly dilated that show no marked rapidity of action, and there may be hearts of normal size which may for a long time beat with great rapidity. Apart from the neurotic cases, it might be assumed that an intoxication of the heart, or a deficiency in some

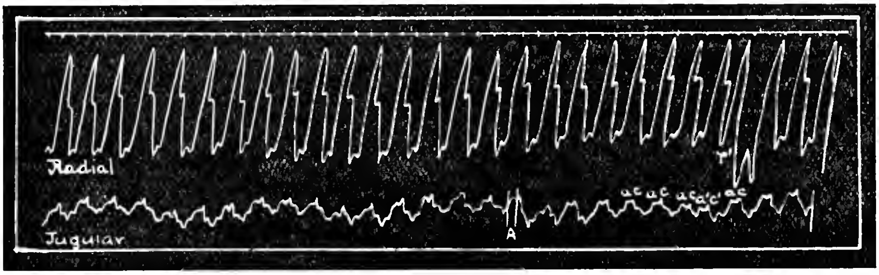

Fig. 71. Simultaneous tracings of the radial and jugular pulses. The rhythm is normal, the rate 164 per minute. There is an auricular extra-systole (a' and c' in the jugular tracing and r' in the radial).

nutriment, is the fundamental cause that renders the tissues more irritable. That the whole tissue is involved, and not merely the cardiomotor centre, is demonstrated in many cases by the quicker contraction of the chambers and the accelerated conduction of the stimulus from auricle to ventricle. Thus, in Fig. 71 the tracing shows a very minute a–c interval, while notwithstanding the abnormal rapidity of the heart's action, the excitability of the auricular muscle was so great that a premature auricular systole actually appeared.

§ 131. **Prognosis.**—A number of people whose hearts beat too frequently show no sign of heart trouble. We then can gauge their condition by their reserve force. Apart from cases with a previous rheumatic history, or of serious heart mischief, I have found that people with continuous rapid hearts gradually recover so far as the heart's condition is concerned, and even cases of exophthalmic goitre may gradually recover with the heart slowing down. If an alcoholic will but mend his ways before

he has induced organic changes in his other organs, the heart shows a wonderful power of recovery. Manifestly, in the other ailments, as tubercular and malignant diseases, the future progress of the case is to a certain extent independent of the heart affection. I do not like the continued rapidity in cases with valvular lesions, as it implies a serious impairment of the myocardium, and if they do not respond to treatment they generally speed on to a fatal issue.

CHAPTER XVI

Diminished Frequency of the Heart's Action

§ **132. Definition of the term 'bradycardia'.**—The term 'bradycardia' has been used when the arterial pulse was slow, and from this it has been inferred that the whole heart was slow in its action. The result of this usage has been to employ the term in many cases quite inappropriately. Thus it is most commonly used in association with the condition known as 'heart-block', a condition, as will be shown later, where the ventricle alone beats slowly, the auricle pursuing a normal or even accelerated rate.

In order to differentiate between the different forms of slow pulse-rate, it is necessary to make observation of the movements of the various chambers of the heart. If this is done, it will be found that the cases of diminished frequency of the *pulse* can be divided into four classes: (1) Those where all the chambers of the heart participate in the slow action (normal bradycardia); (2) where the slow pulse-rate is due to a missed beat, the ventricle having contracted, the resulting pulse-wave being too feeble to reach the wrist (described in § 142, Figs. 85 and 86); (3) certain cases of nodal rhythm where the auricle has ceased to beat, or does so synchronously with the ventricle (nodal bradycardia described in Appendix IV, p. 337); (4) where the stimulus is blocked between auricle and ventricle so that the auricle beats at its normal rhythm, and the ventricle does not respond to the auricular systole, but pursues an independent and slow rhythm (heart-block, described in § 168, Figs. 122 and 123); (5) where the vagus slows the heart, producing standstill of the whole heart for irregular periods (p. 24).

§ **133. Normal bradycardia.**—This only occurs when all the chambers of the heart participate in the slow action. The demonstration of the character of the slowing is best shown by tracings of the jugular pulse with the radial (Fig. 48, p. 113) or apex beat (Fig. 72, p. 139), where the auricle is seen to beat at the same rate as the ventricle. I have never

found this form of slow heart beating under forty per minute, though sometimes we meet with single pauses lasting nearly two seconds (Fig. 79, p. 146). There is a number of people in the enjoyment of perfect health, whose pulse beats regularly about fifty per minute. Those of whom I have kept a record were mostly tall men. In a great many people of spare habit, who suffer also from the X disease (§ 65), the heart-rate may fall under fifty beats per minute. In some of these a rise of temperature of one or two degrees may actually make the pulse beat slower. There are other conditions which may induce a slowing of the heart's pulse, such as increased arterial pressure in Bright's disease, in gout, and in certain cases of arterial

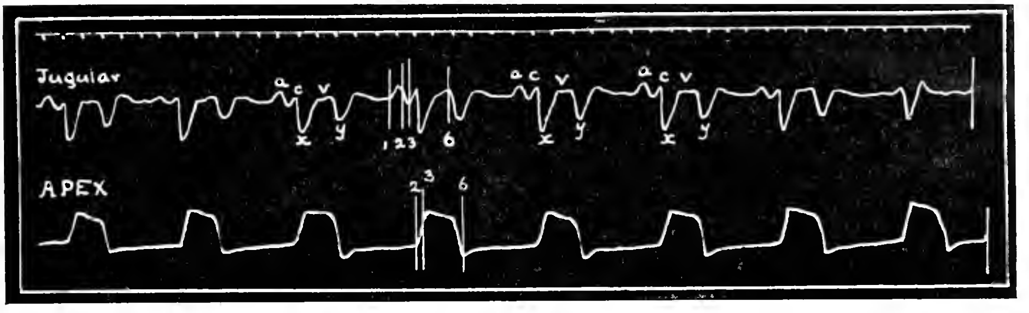

FIG. 72. Simultaneous tracings of the jugular pulse and apex beat, showing the participation of auricle and ventricles in true bradycardia. Rate 50 per minute.

degeneration. In pregnancy the pulse may also be occasionally slow. Jaundice is said to have a considerable power in slowing the pulse, but I myself have never found it.

Occasionally we find patients losing their memory, and the pulse will be found to be very slow—between forty and fifty beats per minute. Further, certain phases of respiration may slow the pulse, and also the exposure of the body to the cold air or to cold baths. So far as I have observed, I have never found any serious result from such slowing of the heart, and I have watched patients whose pulse may frequently be found about fifty per minute, for fifteen and twenty years.

CHAPTER XVII

The Irregular Action of the Heart

Irregular action of the heart is of importance in indicating the mechanism of many of the heart's actions, and a knowledge of this mechanism is essential to the proper diagnosis of pathological changes. As it is a subject of some complexity, I give in this chapter a brief review of the more important points bearing upon irregular rhythm, and a classification of the more common forms.

Irregularities are of such great frequency, and their presence so readily recognized by both patient and physician, that it is necessary clearly to recognize their meaning and significance. Until a few years ago their nature was shrouded in obscurity, and in consequence the mystery regarding them in a great measure oppressed both patient and physician. The fact that in some instances irregularity was of serious import led to the supposition that all irregularities are signs of some grave mischief. In consequence of this many patients are subjected to unnecessary fears, made to carry out elaborate methods of treatment, and have imposed upon them burdensome and unnecessary restrictions.

The advance that has been made in the knowledge of this subject within recent years positively constitutes a revolution. By the combined efforts of clinicians and experimental physiologists, what was recently a complete mystery is now one of the best understood matters in the whole science of medicine. Not only has the scientific aspect been followed out thoroughly, but by watching individual cases for years and noticing the changes that have taken place with advancing years, and observing how people with irregular hearts have borne the stress of life, I have endeavoured to obtain a clearer conception of the bearing of the different irregularities upon the future history of the patient.

§ 134. **Places where the heart's contraction may start.**—The starting-place of the heart's contraction is in the remains of the sinus-venosus that have been incorporated in the veins, such as the node of tissue described

by Keith and Flack at the mouth of the superior vena-cava (1, Fig. 2, p. 15). In describing the functions of the primitive cardiac tube (of which the above-mentioned node is a portion) it was pointed out that any part of the structure was capable of starting the contraction, and that it was because the venous end was the more excitable that the normal rhythm started there (p. 12). When another part of this primitive tube becomes from any cause more excitable than the sinus portion, then the contraction starts at that more excitable part, and an abnormal rhythm results. If a break should occur in the extension of the primitive tissue, the two divisions of the heart will beat separately and independently, as is shown in the Stannius' ligature (p. 13). In what is called 'heart-block' such a separation occurs, and auricle and ventricle beat at independent rhythms. For practical purposes, we can therefore reasonably assume four places where the contraction of the human heart can start :—

(*a*) At the mouth of the great veins where the remains of the sinus venosus still persist, giving rise to the normal or sinus rhythm. (*b*) At the a.-v. node, where auricle and ventricle contract simultaneously. (*c*) In the a.-v. bundle on the ventricular side of the a.-v. node, where, while the auricle contracts in obedience to the sinus rhythm, the ventricle contracts independently. (*d*) In the auricular tissue, where some part of the primitive tube seems to persist.

§ 135. Classification of irregularities.

(1) *Sinus irregularities.*—The heart's contraction arising normally in the remains of the sinus venosus is set to a regular rhythm. The sinus tissue may be excited or depressed as by nerve influence, and irregularities may then occur. This form of irregularity is characterized by a varying length of the cardiac cycle, mainly of the diastolic portion, the pulse beats being always of equal size or nearly equal size, and presenting no 'imperfect systoles' or 'missed beats'. The variation usually corresponds with certain phases of respiration. It is most frequent in the young, but is occasionally present in adult life (see Chapter XVIII).

(2) *Extra-systoles.*—Here an auricular or ventricular systole, or both together, may start prematurely and independently of the sinus rhythm. They occur occasionally in an otherwise regular heart; a premature beat of the radial pulse is felt, followed by a long pause, or there may simply be a long pause (intermittent pulse). Sometimes these extra-systoles may occur with greater frequency, even every second beat being of this nature (pulsus bigeminus). When they are so small as to be imperceptible to the finger, it might seem as if the heart were beating extremely slowly. On auscultation synchronous with the premature beat, two short, sharp sounds are

heard—the first and second sound of the premature or extra-systolic contraction. These sounds are very characteristic of this condition (Chapter XIX).

(3) *Nodal rhythm.*—In advanced disease of the heart from rheumatism and cardio-sclerosis, the starting-place of the contraction is no longer at the sinus, but in some part lower down, where the auricle and ventricle are stimulated to contract for the most part simultaneously. Beats of varying size follow one another at varying intervals ; sometimes the irregularity is extreme, sometimes scarcely perceptible, but careful analysis will usually show variations in the length of the cardiac cycle. This irregularity is usually associated with marked diminution of the heart's power, sometimes extreme, at other times only indicated by a limitation of the field of cardiac response when the patient makes an effort. It may occur at all ages. The heart's rate is as a rule more frequent than normal and it may be extremely rapid temporarily (paroxysmal tachycardia) or continuously ; when continuously rapid, it may slow down and beat about seventy to ninety per minute. In some cases it is less frequent than normal (nodal bradycardia, p. 337).

(4) *Irregularities due to failure of the conducting power of the primitive bundle.*—This is due to the ventricular systole dropping out in consequence of the stimulus for contraction not reaching the ventricle. This condition is rare, but may occasionally occur in influenza and other infectious complaints, and in old and recent rheumatic hearts, especially after digitalis, and in cardio-sclerosis. A more extreme form of the condition is known as heart-block. This condition may be suspected when there is a complete pause in the radial pulse with absence of heart sounds (Chapter XXI).

(5) *Depression of contractility* (*pulsus alternans*).—Irregularities due to the failure of the contractile power of the ventricle are usually regular in rhythm—the beats varying in strength only. The most common form is that where a strong beat alternates with a weak, the rate being quite regular (Chapter XXII, § 179).

CHAPTER XVIII

Sinus Irregularities

§ 136. Character of the irregularity.—As the primitive cardiac tissue at the mouth of the great veins possesses in a degree higher than any other part the power of rhythmically producing the stimulus for contraction, the rhythm of the whole heart follows normally the time set by this portion of the primitive tissue. While normally this rhythm is a fairly regular one, as a matter of observation we find a great many people who show a variation, sometimes slight, sometimes marked, in the duration

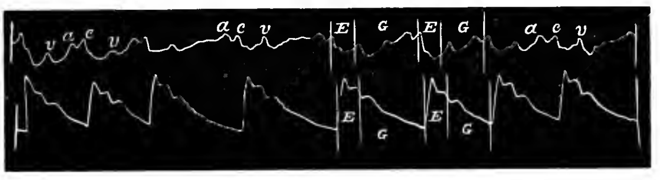

Fig. 73. Simultaneous tracings of the jugular and radial pulses, showing the agreement in rhythm of the right auricle and ventricle (waves *a* and *v*) with the radial pulse, in the sinus form of irregularity. The irregularity is seen to be due to variations in the length of the diastolic period (spaces *G*).

of the cardiac cycle. There is a much greater constancy in the duration of the systolic period of the cardiac cycle than of the diastolic. With the quickening of the pulse-rate, the shortening of the period of the cardiac cycle takes place almost entirely at the expense of the diastolic portion. In sinus irregularities it is the variation in the length of the diastolic period that is the chief characteristic. In the quickened pulse we find the duration of the diastolic period reduced, so that with increase of rate this irregularity disappears. On the other hand, when the heart gradually slows in its action this form of irregularity is prone to occur, so that we find it best in the young and in some adults after a febrile attack or during slow respiration. Typical instances of sinus irregularity are given in Figs. 73 and 74. In Fig. 73

the irregularity is seen to be due to variations in the length of the diastole of the heart (period *G*), the systolic period (*E*) remaining constant. The jugular tracings show that the right auricle (*a*) and ventricle (*v*) participate in the same irregularity as the radial pulse.

§ 137. **Etiology.**—It is generally agreed that this irregularity is of vagus origin. Normally there is a certain degree of inhibition maintained by this nerve, but its centre may become unusually susceptible to impulses from other parts, and these are transmitted reflexly to the heart. This is well seen in some cases where the vagus is more excitable. In certain cases the reflex stimulation of the vagus will produce an alteration of the heart's rate, as in Fig. 75, Plate I, where it is shown that the act of swallowing quickened the heart-rate for a few beats, and then caused a slowing. A more striking illustration of the reflex effect of swallowing on the heart is found in Figs. 258 and 259, Plate IV, where it not only slowed the sinus

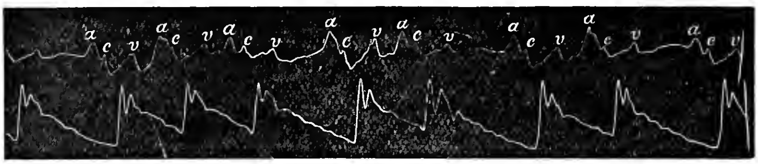

Fig. 74. Simultaneous tracings of the jugular and radial pulses, showing that the auricle participates in the irregularity, and that there is no premature contraction during the long pauses (sinus irregularity).

rhythm, but depressed the conductivity of the a.-v. fibres, so that the stimulus from auricle to ventricle was occasionally blocked. In Fig. 76, Plate I, a vagus effect is shown after the patient has taken three deep and hurried respirations. This patient was under the influence of digitalis, but here the effect is purely upon the sinus, showing a slowing of the rate. In these tracings it is further to be noted that the vagus effect is not immediately produced and does not at once pass off, but lasts some little time. Thus, in Figs. 75, Plate I, 258 and 259, Plate IV, there is shown a secondary slowing some seconds after the swallowing, and in Fig. 76, Plate I, the slowing comes gradually on after the hurried respirations. The reason I dwell upon this is because this sinus arrhythmia is often distinctly respiratory in origin, though the pulse variations do not always correspond with identical phases of the respiration, as in Fig. 77, Plate II, where the slowing occurs at different phases of the respiratory movement.

In the dog this irregularity is very common, and disappears on section of the vagus. An irregularity identical with Fig. 79, due to vagus stimulation, is shown in Fig. 78.

§ 138. **Symptoms.**—This irregularity is easily recognized. To the finger the pulse-rate is continually changing, usually with respiration, and the beats are equal in strength. On auscultation the sounds are heard clear and distinct, and the interval between the first and second sounds is constant. By the ear, the varying difference in the diastolic period can be made out more easily than by the pulse. In rare instances the slowing may occur at rare intervals and affect only one or two beats, as in Fig. 79, and there may be at first some difficulty in recognizing the nature of the irregularity, but when the condition of the patient in other

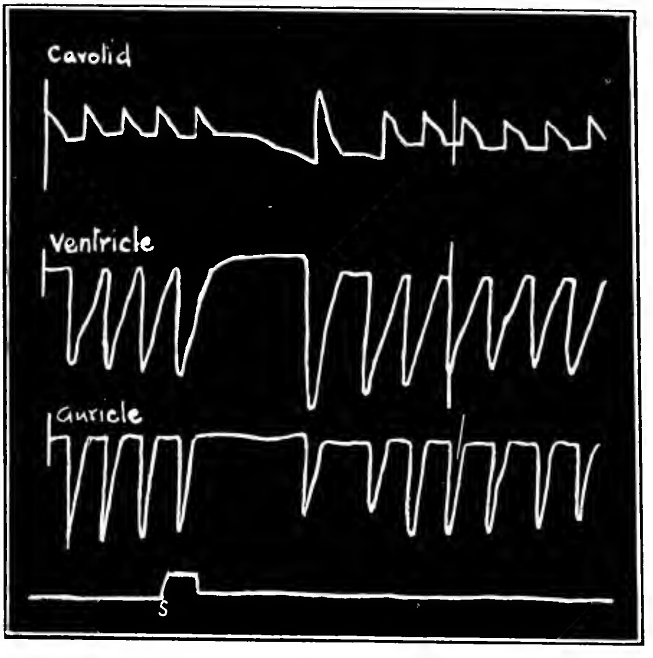

FIG. 78. Shows the effect of vagus stimulation in the dog's heart. The downward movements in the tracing of auricle and ventricle are due to the systole. The vagus was stimulated at *s*, and produced a standstill of the whole heart (Cushny).

respects is taken into account, the nature of the irregularity can be inferred with certainty. Such an irregularity from any other cause would show evidence of severe heart trouble (as heart-block or the nodal rhythm), whereas in such cases there is no evidence of heart trouble, or but the very slightest. Tracings of the jugular pulse at once determine the nature of the irregularity, by showing that the auricle is also subject to the same influence.

§ 139. **Associated symptoms.**—These are merely incidental, the

irregularity itself causing no subjective symptom. When some incidental phenomenon such as syncope appears, an undue importance may be attached by the physician to the irregularity. Many young folks have syncopal attacks, and this irregularity, being the only abnormal feature found by the physician, is often the ground on which unnecessary alarm and unnecessary treatment are based.

Although in the vast majority of cases the slowing of the heart, presumably due to vagus action, gives rise to no symptoms, particularly in the young and when of respiratory origin, yet the period of standstill may be at times so long as to produce an effect on the brain. The patient from whom Fig. 79 was taken had attacks of giddiness at times, and the pauses in the heart's action were often longer

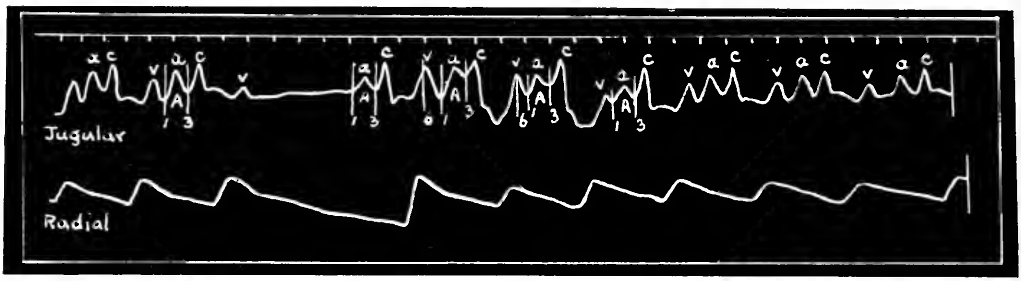

FIG. 79. Occasional slowing of the whole heart due to inhibitory nerve influences on the sinus. The *a–c* interval (space *A*) is not affected by the variations in rate. (Compare with Fig. 78.)

than that figured here. Laslett [308] records a case and gives numerous tracings in which the standstill was frequently so long that the patient lost consciousness.

In the vague condition which I have described as the X disease (§ 65) the respirations are often slow, falling sometimes to seven per minute. When this occurs, the pulse invariably shows this irregularity (Fig. 77, Plate II). It has also been found present in neurasthenia, and some go so far as to diagnose neurasthenia by this irregularity alone. Nicholson [214] has demonstrated its presence in infants, Watson Williams [230] in healthy schoolboys, and Deane [191] in athletic soldiers, and I have found it in a great number of healthy individuals, so that one can safely look upon its association with neurasthenia as merely incidental. It is this form of irregularity which is present in tubercular meningitis.

§ **140. Prognosis.**—There is no reason for attaching importance to this irrĕgularity, and no condition should be considered more grave because of its presence. After a febrile attack it may usually be looked

upon as a favourable sign. Its presence in tubercular meningitis adds nothing to the seriousness of the condition.

The presence of this sinus irregularity may even afford grounds for a favourable prognosis when it occurs in the young after recovery from rheumatic fever, even though there may be a mitral systolic murmur, for it is not present when there is exhaustion of the heart muscle.

Treatment.—It calls for no special treatment, nor should any attempt be made to treat this symptom alone.

CHAPTER XIX

The Extra-systole

§ **141. Definition of the term ' extra-systole '.**—There are so many conditions that simulate extra-systoles, that a good deal of confusion exists in regard to what really constitutes an extra-systole, and it is therefore necessary to define the term. As the stimulus for contraction arises normally in the remains of the sinus venosus at the mouths of the great veins, and as the stimulus passes from these places to the auricle, then to the ventricle,

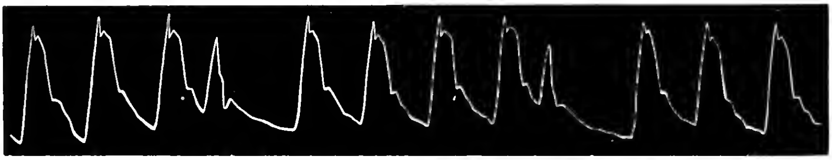

Fig. 80. The small beats are due to extra-systoles.

so that there is normally a sequence of stimulation and contraction of sinus, auricle, and ventricle, I would suggest that the term ' extra-systole ' should be limited to those premature contractions of auricle or ventricle in response to a stimulus from some abnormal point of the heart, but where otherwise the fundamental or sinus rhythm of the heart is maintained.

§ **142. The character of the irregularity.**—The extra-systole is usually recognized by the occurrence of a premature beat in the radial pulse followed by an abnormally long pause, as is shown in Fig. 80, where

the two small beats are extra-systoles. It may appear only at rare intervals, or it may occur at frequent irregular intervals, or regularly after every 1, 2, 3, 4, or more, normal beats, as in Figs. 81, 82, 83, 84.

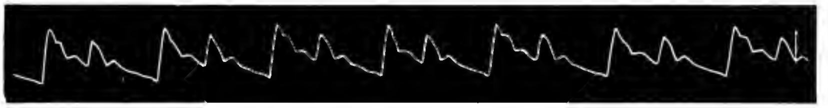

FIG. 81. Pulsus bigeminus due to an extra-systole occurring after each normal beat.

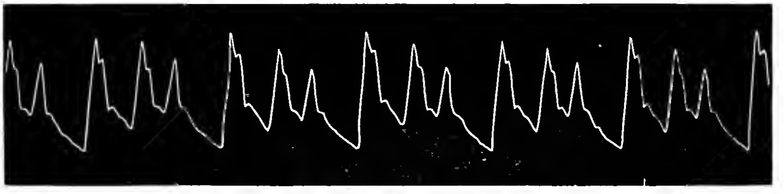

FIG. 82. Extra-systole occurring after every two normal beats.

FIG. 83. Extra-systoles occurring after every three normal beats.

FIG. 84. Extra-systoles occurring after every four normal beats.

The ventricular contraction causing the extra-systole may be so weak that no wave is perceptible to the finger in the radial, though it may be detected in the sphygmogram, as in Fig. 85. In some cases it may even not appear in the sphygmogram, but the heart's sounds, or a tracing of the apex beat at the same time, show that during the long pause in the radial pulse the ventricle contracted, but not with sufficient strength to send a wave into the radial artery (Fig. 86).

In these cases the pulse is described as 'intermittent', or, if the extra-

systoles occur regularly after each normal beat, the pulse at the wrist appears extremely slow, and the case may be put down as ‘ bradycardia ’, or heart-block. It may be differentiated by the observation of the jugular pulse or apex beat, or by auscultation.

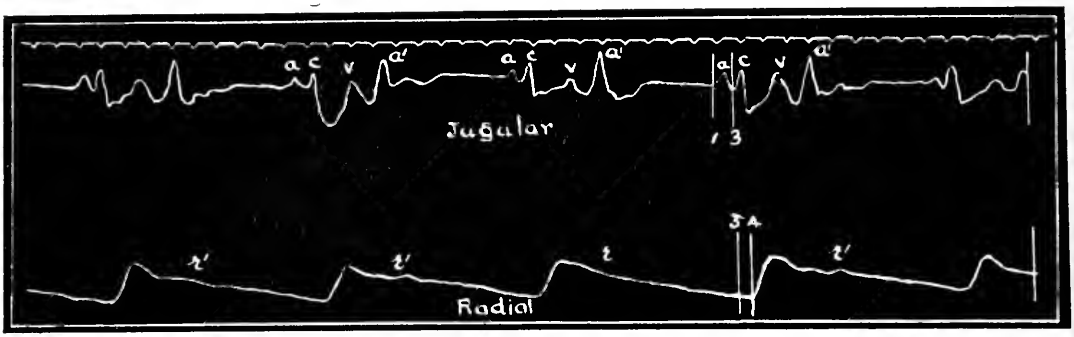

FIG. 85. Slow pulse feeling to the finger about 30 per minute. The small waves (r') are due to extra-systoles and were not perceptible to the finger.

The extra-systole is easily recognized on auscultation. The regular sequence of sounds is interrupted by two short, sharp sounds (if very feeble,

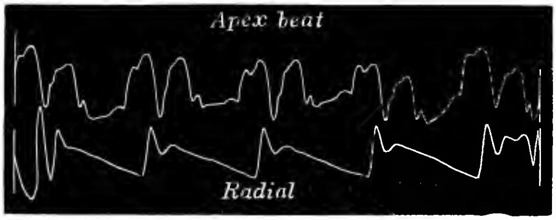

FIG. 86. Simultaneous tracings of the apex beat and of the radial pulse. There are two beats of the apex to one radial pulse.

only one sound may be heard) followed by a long pause, as is represented in the diagrams, Figs. 87 and 88.

FIG. 87. Diagram representing the sounds of the heart with occasional extra-systoles.

§ 143. **Etiology.**—In seeking for an explanation for the occurrence of extra-systoles, I collected a large number of instances and then sought for some basis of classification. Hitherto the classification has to a great extent been based on the results of experiments, but I found in the human

heart many variations which did not coincide with experimental results. After trying various methods, I took up the idea that the remains of the primitive cardiac tube in the human heart might preserve the functional peculiarities of the original tube. One of these peculiarities is the power possessed by any part of the tube, if rendered more excitable, to start the heart's contraction before the sinus. As the primitive tube in the heart is represented at the mouths of the great veins, in the auricle, in the a.-v. node, and in the a.-v. bundle after it leaves the node (Fig. 2), I reasoned that while the normal rhythm started at the veins, premature contractions might start in the remains in the auricle, a.-v. node, or bundle. Examining my tracings with this view in mind, I found that the vast majority readily fell into this classification.

A further proof was found in the fact that in many cases the power of conduction in the a.-v. bundle was impaired, implying an invasion of this system by the disease, a view further supported by post-mortem examinations.

FIG. 88. Diagrammatic representation of the sounds of the heart in a case of such rhythmical irregularity as is represented in Figs. 81, 85, and 86.

While I do not assert that this view is proven to be correct, I use it, as it brings out more clearly the salient points of the varied forms of extra-systole. I claim no originality for this suggestion, as the possibility of arrhythmias arising at the a.-v. bundle has occurred to many people.

§ 144. **Ventricular extra-systole.**—(The place of origin of ventricular extra-systoles is assumed to be in the a.-v. bundle beyond the a.-v. node or in some part of its branches in the ventricular wall.) The simplest form of extra-systole is that where a premature contraction of the ventricle is interpolated between two normal beats.

In Fig. 89 there are two small waves *r′* in the radial, which are due to extra-systoles of the left ventricle. In the tracing from the neck the waves *c* are due to the carotid, the waves *a* are due to the auricle, and the waves *c′* are due to the extra-systoles and correspond to *r′* in the radial. The diagram intercalated between the radial tracing and the tracing from the neck shows the relationship of the different events. The downstrokes in the upper compartment represent the beginning of the auricular systoles, and the downstrokes in the lowest compartment represent the beginning of the

carotid pulse beats, while the slanting lines in the middle compartment represent the time between the beginning of the auricular and carotid waves (the *a–c* interval). In the cardiac cycles, apart from those in which the extra-systoles *r′* and *c′* occur, the ventricular contraction follows regularly on the auricular, and it is assumed that the sinus rhythm governs the contraction of the auricle. The extra-ventricular contractions are seen to start independently of the auricular contraction—that is, they are extra-systoles of the ventricle. The same features are to be recognized in Fig. 90, Plate II, where there are four extra-systoles (*r′* in the radial, and *c′* in the jugular tracing); it will be seen that the auricular wave appears at regular

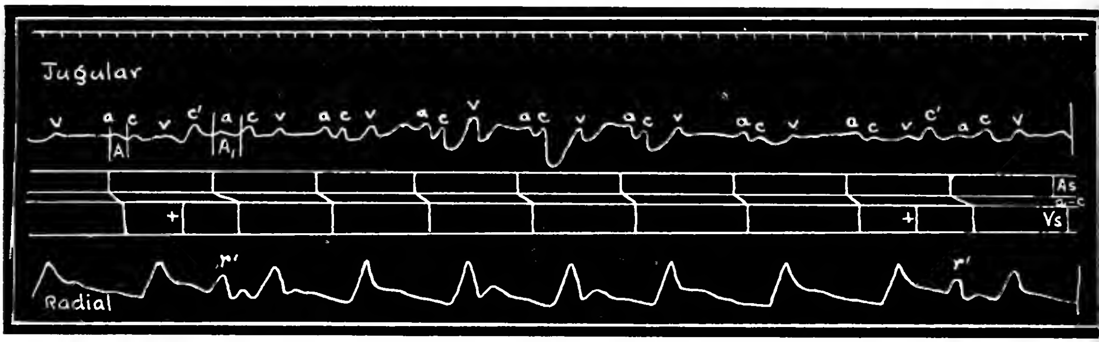

FIG. 89. Simultaneous tracing of the jugular and radial pulses, showing the interpolation of ventricular extra-systoles (*c′*, *c′* and *r′*, *r′*) represented in the diagram by the downstrokes + +. The downstrokes in the space *As* represent the auricular waves (*a*) in the jugular, and the downstrokes in the space *Vs* represent the carotid waves *c*, and the slanting lines connecting them represent the *a–c* interval.

intervals, and that the extra-systoles are interpolated between two normal beats.

In Fig. 91, Plate II, there is an extra-systole during each of the irregular periods *B* and *C*. In the jugular tracing the carotid wave *c* follows the auricular wave *a*, but during the two irregular periods the carotid wave *c′* precedes the auricular wave *a′*, a fact brought out clearly in the intercalated diagram. We can prove that *c′* is due to the carotid, because it occurs at the same time after the preceding carotid wave that the radial beat *r′* appears after the preceding radial beat. The auricular wave *a′* appears at the normal auricular interval, and the auricle pursues its wonted regular rhythm while the ventricle is contracting prematurely and independently.

In the irregular period *B* the interval between *a′* and the following *c* is greatly lengthened (space A_1). The reason of this delay is shown in the diagram to be due to slower passage of the stimulus from auricle to ventricle

—a fact of great importance, which will be dealt with in discussing the condition of the conductivity of the auriculo-ventricular bundle.

In the irregular period C, there is a much longer pause in the radial tracing after the extra-systole r', and the intercalated diagram shows that this is due to the fact that the ventricle does not respond to a stimulus after a'. The difference between the irregular periods B and C is seen, therefore, to be due to the fact that in the irregular period B the ventricle responds to the stimulus from the auricular systole after the extra-systole, while in the irregular period C the ventricle does not respond.

The irregular period C represents the most common form of extra-systole, and its appearance in graphic records is usually easily recognized by the presence of the long pause after the premature beat r'.

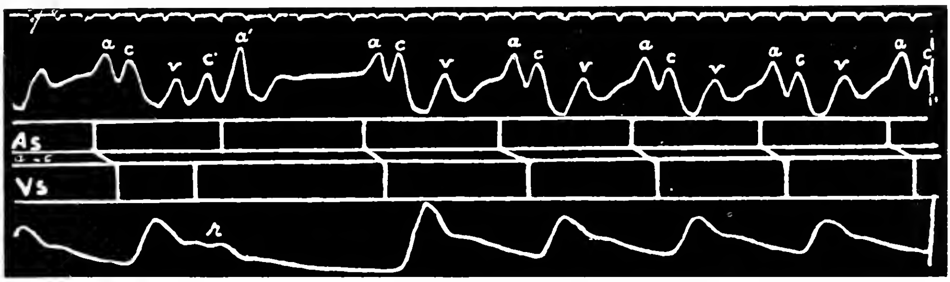

Fig. 92. Represents a common form of ventricular extra-systole. There is a long pause after the extra-systole (r'). In the jugular tracing and in the diagram this is seen to be due to the failure of the stimulus from the auricle (a') to provoke a ventricular contraction.

Thus Fig. 92 is a very characteristic example, and the intercalated diagram shows a regularly acting auricle and the premature and independent ventricular contraction with no response to the auricular systole after the premature ventricular contraction, which accounts for the long pause.

Simultaneous occurrence of the normal auricular systole and of the ventricular extra-systole.—In the illustrations I have given of the ventricular extra-systole (as in Figs. 89 and 92), in the jugular tracing the carotid wave c' was seen to precede the auricular wave a'. In these instances the normal rate was rather slow. In most instances, however, they fall together—the auricular contraction occurring during the ventricular contraction. In consequence of this the auricle cannot empty its contents into the ventricle, and hence a big wave is sometimes sent into the jugular. In patients with a well-marked jugular pulse, this is readily recognized by the eye. In other cases the jugular pulsation is only to be seen when this big wave is sent back.

In Fig. 93 the waves *a* and *a′* are due to the auricular systole, and occur at regular intervals. The waves *a′* are, however, much larger than the waves *a*, and the reason for the increase in size is found in the fact that at that time the ventricle was also in systole, causing the extra-systole. In Fig. 94 a simultaneous record of the apex beat and jugular pulse shows the premature contraction of the ventricle *o* at the same time as the large

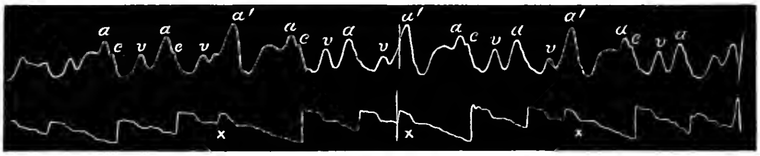

Fig. 93. Simultaneous tracings of the jugular and radial pulses. The small beats × × × are extra-systoles. The auricle preserves its rhythm during the irregular periods in the radial pulse. The wave *a′* is the auricular wave during the premature contraction of the left ventricle. The absence of the ventricular wave *v*, after the wave *a′*, indicates that the right ventricle had contracted early, evidently synchronous with the premature contraction of the left ventricle, the large wave following *a′* being due to stasis.

auricular wave *a′*, so that here also we have evidence of the simultaneous contraction of auricle and ventricle during a ventricular extra-systole. When there is a large jugular pulse, the increased size of the auricular wave occurring during the extra-systole of the ventricle may not be so marked as in Fig. 95.

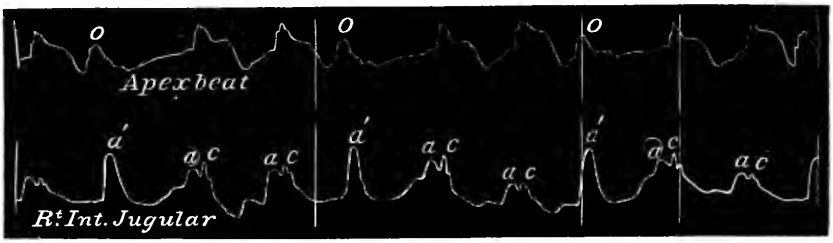

Fig. 94. Simultaneous tracings of the apex beat and of the jugular pulse, showing the rhythmical appearance of the auricular wave during the irregular periods in the apex tracing. The small beats *o o o* are extra-systoles of the ventricle.

§ 145. **The auricular extra-systole.**—(The suggestion put forward here is that, in the production of the auricular extra-systole the stimulus arises in the primitive tissue incorporated in the auricle—the exact site being still undetermined.)

When an extra-systole arises in the auricle, the sounds of the heart and the radial tracing present exactly the same features as when a ventricular extra-systole occurs, and it is only by a simultaneous record of the jugular

pulse that the one can be differentiated from the other. In Figs. 96 and 97 the radial pulse shows premature beats (r'), which can be readily identified as extra-systoles.

In the jugular tracings the carotid wave c' is preceded by an auricular wave a', and the only inference that can be drawn is that a' is due to an

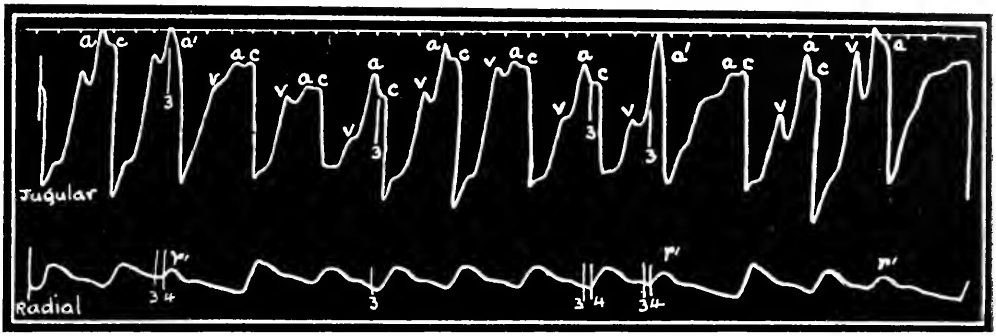

FIG. 95. Shows three ventricular extra-systoles in the radial (r') while the auricle pursues its regular rhythm (a and a'). The downstrokes 3 and 4 represent the time of the carotid and radial pulses.

extra-systole of the auricle, which is followed by an extra-systole of the ventricle, producing the carotid and radial beats c' and r'.

In such instances as these the auricle is supposed to contract in reply to a stimulus from some source other than the sinus, and the long pause after the extra-systole is due to the fact that the stimulus from the sinus arising at

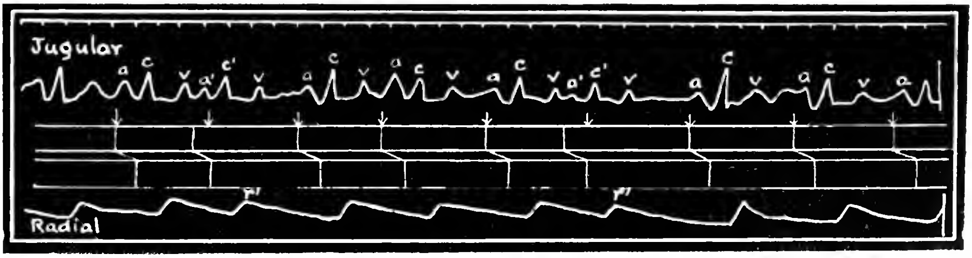

FIG. 96. Shows auricular extra-systoles (a') followed by ventricular contractions (c', r'). The arrows in the diagram represent the sinus stimulation and the long pauses after the extra-systoles are seen to be due to the fact that the auricle did not respond to the sinus stimulation.

its normal time fails to provoke the refractory auricle to contraction. This is brought out in the intercalated diagram in Fig. 96, in which, above the upper space, are arrows representing the sinus stimulation. In the diagram it will be seen that there is no response to the sinus stimulation after the extra-systole, but that auricle and ventricle remain quiescent till the following

sinus stimulation excites them to a contraction. In these two illustrations the irregular period is equal to two cardiac cycles, the explanation being obvious from the study of the diagram in Fig. 96. In most cases of auricular extra-systole, the irregular period is less than two cycles, as is shown in Figs. 98 and 99. The reason given by Cushny [186] and Wenckebach [228], and usually

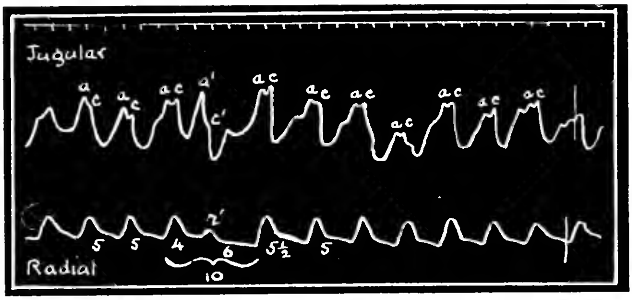

FIG. 97. Shows an auricular extra-systole a'. The numbers represent tenths of a second and the irregular period (r') equals two normal periods.

accepted, is that the stimulus arising in the auricle passes back to the sinus and stimulates the sinus so that its stored energy is exhausted, and it begins to build up anew the stimulus material. As soon as it has again reached the excitable stage, it starts off the contraction. Thus in the diagram

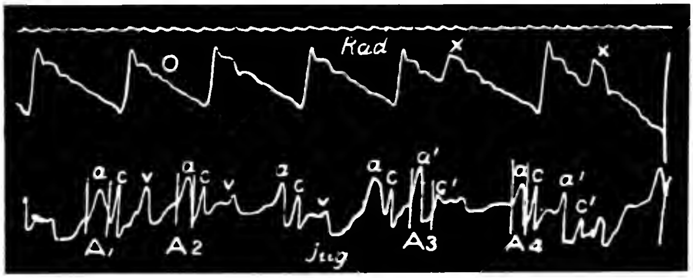

FIG. 98. Shows two premature or extra-systoles of auricular origin (×). The waves c' in the jugular tracing occur at the same time as the small premature beats (+) in the radial tracing, and are therefore due to the carotid. These are preceded by premature waves a' due to the auricle. The interval a'–c' (space A_3) is greater than the average a–c interval (A_2), and is much greater than the following a–c interval (A_4).

(Fig. 100), representing the events in Fig. 99, the stimulus is represented coming down from the sinus to the auricle, but at the extra-systole (+) the stimulus is represented by an arrow passing back to the sinus, so that the sinus responds to the retrograde stimulation. After this premature stimulation, the sinus rests for a normal period, and starts off again at the normal

rhythm. This irregular period, due to the auricular extra-systole, is so frequently shorter than two normal beats that this is usually assumed to be the manner in which it is brought about. While it offers a plausible explanation, it cannot be said in any sense to have been proved, and there are other possibilities which need consideration before it can be finally accepted.

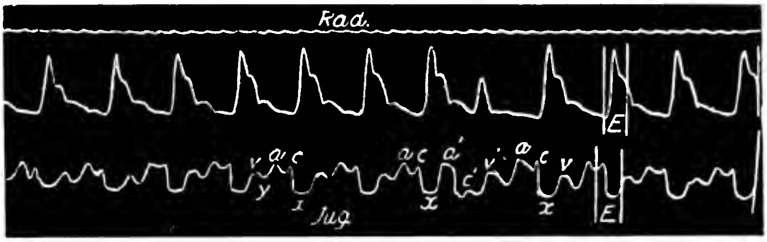

FIG. 99. Shows an extra-systole of auricular origin at a'. This tracing is interpreted in the diagram, Fig. 100.

As, however, they are still speculative, it would not be convenient to discuss them here.

§ 146. The extra-systoles arising in the auriculo-ventricular node (nodal extra-systole).—So far the recognition of extra-systoles as ventricular or auricular has been comparatively easy. There is a third class which has hitherto not been sufficiently considered, and which, in my opinion, has been wrongly interpreted. The characteristic feature of these

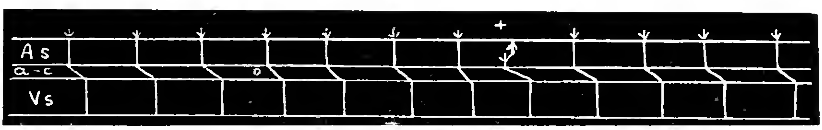

FIG. 100. Diagram representing the events in Fig. 99. The extra-stimulation at + is represented arising in the auricle, passing back and disturbing the sinus rhythm. Note the lengthened a–c interval after the auricular extra-systole.

extra-systoles is that auricle and ventricle contract prematurely and together. In Figs. 93, 94, and 95, it was demonstrated that auricle and ventricle contracted together when the ventricular extra-systole appeared at the same time as the normal auricular systole. In these cases the auricle could be demonstrated to contract at its normal period. In another class of cases it can be demonstrated that both auricle and ventricle contract prematurely and together, the auricular wave appearing at the same time as the arterial

pulse. Thus in Figs. 101 and 102, the auricular waves a' in the jugular appear prematurely, and obscure the appearance of the carotid waves,—the time when the latter were due can be ascertained by measuring the time between the extra-systole in the radial pulse, and the preceding beat. That the auricle did not contract at its normal time is evident by the absence of any wave at the time of the arrow in the intercalated diagram of Fig. 101

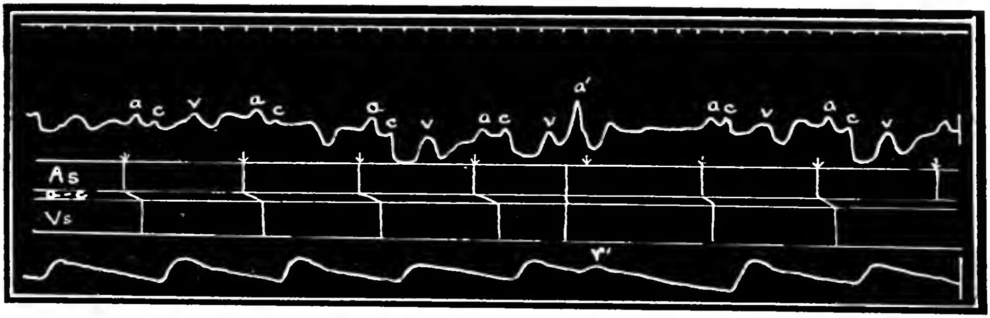

Fig. 101. Shows a nodal extra-systole (a' and r'), the auricular and ventricular systoles as shown in the diagram are premature and simultaneous.

during the irregular period, which is the time when it was due. Here then we have evidence that both auricle and ventricle contracted prematurely and simultaneously. In the ventricular and auricular forms no difficulty is found in recognizing that the extra-stimulation must have affected either one chamber or the other, but in this form we have to consider where a stimulation could arise that would at once affect both chambers. If all the possibilities be considered, we are driven by a process of exclusion to attribute the source of this stimulation to the tissue that joins auricle and ventricle, and almost to a certainty to that portion described as the auriculo-ventricular node (2, Fig. 2). This will be brought out more clearly in the later discussion.

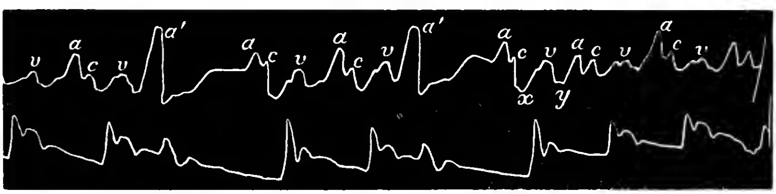

Fig. 102. Shows two nodal extra-systoles (a'), the auricular waves a' appearing prematurely and at the same time as the extra-systole in the radial.

In many cases it is difficult to tell whether the stimulus producing the extra-systole arises in the ventricle at the same time as the normal auricular systole, or whether they have contracted together in response to an abnormal stimulation. In the latter case the abnormal stimulation affects the auricle at the same time that the normal was due, so that we do not get the proof of the premature contraction of the auricle. My reason for this suggestion is that sometimes we meet with a variety of extra-systoles in the same patient, indicating that at one time the auricle is prematurely stimulated, at another time the ventricle, and again both ventricle and auricle together. This is well seen in Fig. 103, Plate II, where three forms of extra-systole occur. The wave a' in the irregular period C is manifestly an auricular extra-systole followed by a premature carotid wave c', which corresponds in time to the extra-systole in the radial tracing. In the irregular period B, the

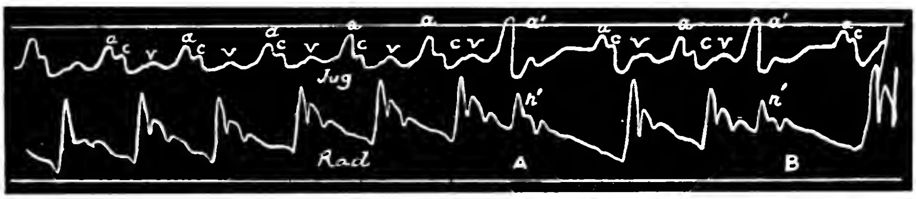

Fig. 105. Shows two nodal extra-systoles. During the irregular period A, the large auricular wave a' appears at the normal auricular period, while during the irregular period B, a' and r' occur prematurely and synchronously.

auricular wave a' appears at its normal interval, but in the radial tracing there is seen an extra-systole which appears at the same time as the wave a'. This is undoubtedly a ventricular extra-systole. In the irregular periods A and D, the conditions are not the same. The wave a' here has quite a different appearance from that in either B or C, being broader at the top. It is difficult to be certain whether it appears before the normal auricular systole was due, as there is a little sinus arrhythmia—the pulse periods not being of equal duration. The absence of the extra-systole in the radial does not imply that the left ventricle did not contract, for, as we shall see later, the ventricle may contract so feebly that a pulse-wave does not reach the wrist. That they are probably nodal extra-systoles can be inferred from the nature of the extra-systoles that occurred from time to time in the patient. Thus the typical auricular extra-systole in Fig. 99 is from the same patient. Fig. 104, Plate II, is also from the same patient, and shows two irregular periods in which the large wave a' appears before a normal auricular systole is due.

From this I suggest that this wave is of the same nature as that of *a′* in the irregular periods *A* and *D*, Fig. 103, Plate II, namely, due to a premature and simultaneous contraction of auricle and ventricle. The suggestion that the abnormal stimulation may affect auricle and ventricle at the time the normal auricular stimulus is due is supported by such a tracing as Fig. 105. There are here two irregular periods, *A* and *B*, due to extra-systoles. The extra-systole in *A* is only slightly premature, and the large wave *a′* appears at the time the normal auricular wave was due. The extra-systole in *B* appears earlier, with the result that the large wave *a′* appears before the time the normal auricular systole was due, so that here auricle and ventricle contracted simultaneously and prematurely.

This explanation of the occurrence of the extra-systole arising in the auriculo-ventricular node is not the one usually accepted. It has been assumed that these cases are really ventricular extra-systoles, and that the stimulus has passed back and prematurely stimulated the auricle, ‘ a retrograde extra-systole ’ as it has been called. But that this explanation is not tenable can be proved on several grounds. As was shown in Figs. 89, 90 Plate II, 91 Plate II, and 92, there is no tendency for the stimulation from the ventricular extra-systole to affect the auricular rhythm. If the stimulus does travel back, the rate backward must be much greater than that at which the normal stimulus travels forward, for as shown in the diagram, in Fig. 101, the time of the ventricular systole is exactly at the same time as the auricular. That this time should be shorter is very unlikely for two reasons—(1) the auriculo-ventricular bundle would be stimulated by the ventricular extra-systole so soon after its conductivity had been exercised, that it would not have recovered, and a backward stimulus would therefore be delayed, just as the normal stimulus was delayed after the extra-systole in Fig. 89. (2) The simultaneous and premature contraction occurs in patients in whom the conductivity of the auriculo-ventricular bundle is impaired, as I shall show presently.

§ 147. The condition of the a.-v. bundle in cases showing extra-systoles.—From what I have said, there are three forms of extra-systole all arising demonstrably from different places in the heart. Moreover, certain individuals exhibit all these forms, and it is therefore reasonable to infer that all these are due to some common cause. When all the possibilities are considered, the suggestion that the primitive tissue is the seat is the most plausible, and the question then arises, is there any further evidence to be elicited as to the condition of this primitive tissue ?

The only clinical evidence of the condition of the primitive tissue is to be found in measuring the rate at which the stimulus for contraction passes

along the a.-v. bundle from auricle to ventricle. This rate is found by measuring in jugular tracings the time between the beginning of the auricular wave and the beginning of the carotid wave—the *a-c* interval (between lines 1 and 3 in the tracings of the jugular pulse as in Figs. 47 and 48), as I have called it. This interval is occupied by three events : (1) the systole of the auricle ; (2) the transmission of the stimulus from auricle to ventricle ; (3) a minute portion of time taken up by the interval during which the ventricular pressure is rising before the opening of the aortic valves. As (3) is practically constant, any variation in the duration of the *a–c* interval can be attributed to a variation in the rate of the stimulus conduction in the a.-v. bundle (see § 163). In normal individuals, this period very rarely exceeds one-fifth of a second. In hearts acting too frequently, this interval is usually less, sometimes one-tenth. One may suspect something wrong with the a.-v. bundle when this period is one-fifth of a second when the heart is beating rapidly, for the stimulus that quickens the heart-rate will also quicken the rate of conduction when the a.-v. bundle is intact. In healthy slow-acting hearts, in which there is a variation in the heart's rate, it is found that whether the heart's pauses are short or long, the *a–c* interval preserves a remarkable constancy. The best instance of this is found in the young, and in those healthy adults in whom there is present the sinus form of irregularity. Thus in Fig. 79, p. 146, the long pause is due to a standstill of the whole heart, and is a very typical example of ' sinus irregularity ', probably of vagal origin, and here the constancy of the *a–c* interval (spaces *A*) is very well seen.

On the other hand, if we look at the interval between the auricular extra-systole *a′* and the following carotid wave *c′* in Figs. 98 and 99, we find it markedly increased. This implies that the conductivity in the a.-v. bundle had not recovered from its previous stimulation, and the slowness of recovery is the proof of inefficiency. In cases of ventricular extra-systole, as in Figs. 89, 90 Plate II, and 91 Plate II, and in cases of extra-systole arising at the a.-v. node, as in Fig. 101, we find frequently a distinct shortening of the *a–c* interval after a long period of rest, and a lengthening when the period of rest is short. This is well illustrated in Fig. 106, Plate II, where after the long pause the *a–c* interval becomes less than one-fifth of a second (A_1), but with each succeeding beat it gradually lengthens (A_2 A_3 A_4) until it exceeds one-fifth of a second in duration. In elderly people in whom extra-systoles occur, this variation in the conductivity of the a.-v. bundle is almost always present. I have also met with an increase of the *a–c* interval, and extra-systoles after rheumatic fever.

§ 148. **The dropping out of the beat after the extra-systole.**—In most instances there is a long pause after the extra-systole—the so-called

compensatory pause (pulse intermission). The reason given by Engelmann, and usually accepted, is that the ventricle is so exhausted after the preceding contraction that it is refractory to the following normal stimulus. While this may be the explanation when the extra-systole is produced experimentally, it does not hold good for the spontaneous extra-systole in man. In Figs. 89, p. 152, and 90 Plate II, and in the irregular period *B* of Fig. 91, Plate II, there is no compensatory pause, because, as can be seen from the intercalated diagrams in Figs. 89, and 91 Plate II, the stimulus from the auricular contraction immediately following the extra-systole did get through and stimulated the ventricle. In each case, however, the stimulus took a much longer time to pass from auricle to ventricle. In Fig. 91, Plate II, the stimulus failed to get through during the irregular period *C*, for there is no ventricular response to the auricular contraction after the extra-systole. In these and in many similar instances, it is manifest that the reason the ventricle does not contract in response to the stimulus from the auricle after an extra-systole, is that the stimulus is blocked in its passage over the a.-v. bundle. Hewlett [202] has recently recorded a case in which the ventricle did not respond to the stimulus from an auricular extra-systole, and the same explanation is given by him. From the foregoing considerations, I make the suggestion that the compensatory pause is not due to the ventricle being refractory, but to the a.-v. bundle being refractory and not conveying the stimulus to the ventricle, which therefore stands still till the next physiological stimulus comes down from the auricle.

§ 149. Reasons for attributing the origin of extra-systoles to affection of the remains of the primitive cardiac tube.—(1) An *a priori* reason, in the fact that the primitive tube possesses a greater excitability than the auricular and ventricular tissue, and that therefore an abnormal stimulus is more likely to arise in this tissue.

(2) It satisfactorily accounts for the three forms of extra-systole, especially when in any individual two or more forms arise. It seems more reasonable to assume that the one organ or tissue being degenerated gives rise to an extra stimulus, now at the auricular portion, now at the ventricular portion of the bundle, and now at the a.-v. node, than to say it arises now in the auricle, and now at the ventricle, and again at some other part of the ventricle which propagates a stimulus back to the auricle.

(3) The frequent association of extra-systole with impaired conductivity of the a.-v. bundle implies an affection of the tissue in question. It may be objected that an extra-systole is an evidence of exalted function, while impaired conductivity is an evidence of depressed function. Gaskell has shown that the rate at which a stimulus passes across a bridge of heart

muscle can be retarded by narrowing the bridge over which the stimulus for contraction has to pass. In the cases of associated depressed conductivity and extra-systole, there is destruction of some part of the bundle, while the degenerative process which does this, renders the part more irritable, and we meet with the combination of extra-systoles and heart-block.

(4) These degenerative processes are more common in advanced years, hence it is in the elderly we find extra-systole most frequent. So far as Dr. Keith has examined the hearts of patients I have sent him who showed extra-systoles during life, he has invariably found evidence of degeneration of the a.-v. node and bundle. As, however, there was always present degeneration of the coronary artery and muscular fibres of the ventricle, it does not exclude the possibility of ventricular degeneration giving rise to the extra-stimulus, except for the *a priori* reason already given.

I give these reasons, not that I consider they are conclusive, but as suggesting a possible explanation. The matter, however, awaits further experimental work before it can be finally settled. The explanation given seems to me to be more satisfactory than the one hitherto adopted.

§ 150. **Conditions inducing extra-systoles.**—A condition necessary to the production of an extra-systole is an undue excitability of the remains of the primitive cardiac tube. It is not easy in all cases to understand how this arises. In elderly people there are almost invariably sclerotic changes in the coronary artery, and especially in the branch that supplies the a.-v. node and bundle; and secondary changes are usually present in these structures. I have reasoned, therefore, that these secondary changes increase the excitability of this tissue, so that the stimulus for contraction arises quicker here than at the sinus. A great many facts point to the reasonableness of this interpretation. A normal slow sinus rhythm gives an opportunity for the stimulus to arise elsewhere, and it is in the elderly with a slow pulse that one most often finds extra-systoles. On the other hand, extra-systoles sometimes occur in rapidly-beating hearts, perhaps because, although the sinus is discharging impulses more rapidly than normal, these are not sufficient to exhaust the irritability of some very sensitive point in the primitive tube. But not infrequently when a heart which ordinarily exhibits extra-systoles is accelerated, as in slight fever, the extra-systoles disappear, because the impulses arising from the sinus are now sufficient to exhaust the irritability of the part which ordinarily gives rise to the extra-systole.

Another condition that tends to increase the excitability of this structure is the changes subsequent to rheumatic affections of the heart, for in these cases extra-systoles are often present, and frequently the heart's contraction

starts from the a.-v. node continuously. Digitalis may give rise to them in rheumatic hearts. Dyspeptic and neurotic people are often liable. That other conditions give rise to extra-systoles is also evident from the fact that they may occur in young people in whom there is no rheumatic history and no cardio-sclerosis, and whose after-history reveals no sign of heart trouble.

§ 151. **Sensations produced by extra-systoles.**—Some patients are conscious of a quiet transient fluttering in the chest when an extra-systole occurs; others are aware of the long pause 'as if their heart had stopped'; while others are conscious of the big beat that frequently follows the long pause. So violent is the effect of this after-beat that in neurotic persons it may cause a shock followed by a sense of great exhaustion. Most patients are unconscious of the irregularity due to the extra-systole until their attention is called to it by the medical attendant. Both being ignorant of its origin, and it being characteristic of human nature to associate the unknown with evil, patient and doctor are too often unnecessarily alarmed.

§ 152. **Prognosis.**—The most serious thing about these cases is that the consciousness of having an irregularity sometimes makes a patient introspective and depressed. He keeps feeling his pulse, and communicates his doleful tale whenever he finds a sympathetic ear.

As the process which gives rise to it in elderly people is the same as that which produces the tortuous temporal arteries, no more significance should be attached to the one symptom than to the other. I have followed cases for many years, and watched them pass through seasons of sickness and of stress, and have seen no reason to attach any serious import to this symptom. In rare instances, the heart, from being occasionally irregular, has after many years become continuously irregular for short or long periods, and in a few the permanent establishment of the nodal rhythm has been the means of hastening the end. But this is infrequent, and in cases of cardio-sclerosis has only happened in advanced life, and the patient should on no account be frightened by being warned of the possible occurrence of this unlikely contingency. In younger and neurotic people I have never seen it lead to any bad results. It may appear in serious affections of the heart, as in febrile complaints, but it does not of itself add to the gravity of the condition, though I am not sure that when due to an acute affection of the heart, as in pneumonia and rheumatic fever, it may not be a sign of invasion of the myocardium by the diseased process.

§ 153. **Treatment.**—If the patient is aware of the irregularity he should be assured that there is no cause for alarm. It is useless to attempt

to treat the irregularity itself. If in other respects the patient is well, then there is no need for any special treatment. If the patient be suffering from conditions which seem to promote the irregularity, such as worry, fatigue, dyspepsia, the treatment should be devoted to the removal of the pre-disposing cause. In people with temporary high blood-pressure who show extra-systoles, I find plenty of healthy exercise in the open air specially beneficial, though until they get trained, the extra-systoles may at times become more frequent by the exertion.

CHAPTER XX

The Nodal Rhythm (Continuous Irregularity of the Heart—Paroxysmal Tachycardia)

§ **154. Meaning of the term ‘ nodal rhythm ’.**—The term ‘ nodal rhythm ’ is applied to that action of the heart where the auricles and ventricles contract simultaneously—the ventricular contraction preceding the auricular by about one-tenth of a second in the great majority of cases. From this fact, proved by experimental as well as clinical evidence (§ 115), it is reasoned that the starting-place is no longer at the remains of the sinus, but in the remains of the primitive cardiac tube lower down. It has been established that when the a.-v. bundle has been cut across after it has left the a.-v. node (Fig. 2), the auricle and ventricle beat at independent rates—the ventricular rate being slow (true ventricular rhythm, § 168). In the vast majority of cases of nodal rhythm the heart beats more rapidly than normal, and the auricle and ventricle contract together. From these circumstances I have reasoned that here, as in the case of the nodal extra-systole, the source of the heart's contraction starts at or about the a.-v. node—hence the term ‘ nodal rhythm ’. Whether this may ultimately be found to be the real starting-point or not, the cases described under this term have such well-marked characters that it is imperative that they should be singled out for special recognition. *The nodal rhythm is present in the majority of cases of severe heart failure, and in a great many the immediate breakdown is directly attributable to the inception by the heart of this abnormal rhythm.* When it occurs the heart is at once placed at a great disadvantage in carrying on its work, and the extent of the impairment of the circulation depends on the integrity of the heart muscle.

I have already drawn attention to the outstanding features of these cases, where it was shown that the ventricular venous pulse indicated a change in the starting-place of the heart's contraction, and that all sign of the auricular contraction at the normal period of the cardiac cycle dis-

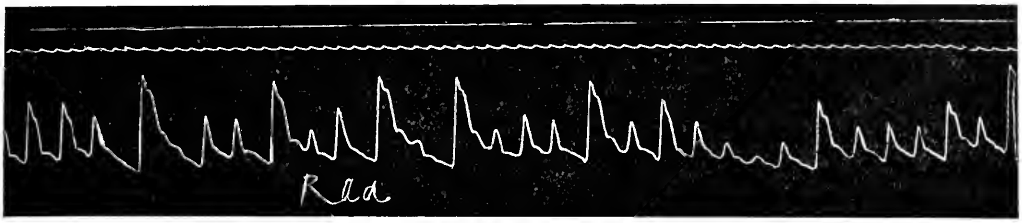

FIG. 107. Tracing of the radial pulse showing the irregularity characteristic of the sudden inception of the nodal rhythm.

appeared. Detailed proof of this is given in the record of illustrative cases cited in Appendix II.

The most obvious sign which, in the great majority of cases, indicates this abnormal rhythm, is the character of the heart's action. Except in

FIG. 108. Shows a type of irregular pulse with the nodal rhythm.

some cases of excessive rapidity, this rhythm is invariably irregular; the irregularity sometimes being extreme. It may be occasionally so slight that it is apt to be overlooked, but if the heart's action be watched for a few minutes, it will show variation in rhythm. The character of the irregularity

FIG. 109. Characteristic type of nodal irregularity occurring in the elderly.

will be better realized by looking at such tracings as Figs. 107, 108, 109, and 110, where the rhythm will be seen to be nondescript and disorderly (other examples will be found in § 115, and in Appendix II).

§ 155. **Etiology.**—The exact pathological details of these cases have not been sufficiently worked out, but from an examination of a series of hearts in which I was able to demonstrate all the features of this irregularity

during life, Dr. Keith has found evidence of pathological changes which, with the well-authenticated clinical evidence, permits the following provisional descriptive account :—

The great majority of cases of nodal rhythm are found among those who have suffered from rheumatic affection of the heart, or cardiosclerosis. In rheumatic hearts there are often deposits of cells scattered through the heart, which ultimately cicatrize. These deposits when on or near the a.-v. bundle impair its function, and this is frequently recognized in early cases by the delay which occurs between the contraction of the auricle and ventricle, from the retarded transmission of the impulse through the connecting fibres. A later result may be obtained when the cicatrization irritates the bundle, and renders it more excitable than the sinus. In accordance with the law that the contraction starts at the most excitable part of the primitive tissue, the contraction of the heart then originates in this more irritable part. Somewhat analogous changes follow in cardio-

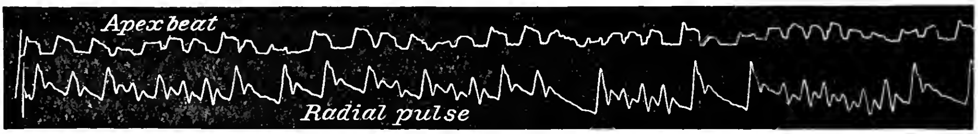

Fig. 110. Simultaneous tracings of the apex beat and of the radial pulse, showing characteristic type of nodal irregularity during a period of severe heart failure.

sclerosis and in degeneration of the coronary arteries. In certain typical cases of this irregularity Keith has found the artery supplying the bundle affected with marked arterio-sclerosis, and with an invasion of fibrous tissue in and around the a.-v. node and bundle (see report of cases in Appendix II).

In these cases the increased irritability of the degenerated a.-v. node and bundle seem to lead to the inception of the abnormal rhythm. Another cause of nodal rhythm is suggested by some cases in which there has been found such distension of the auricle that it appeared impossible that the stimulus from the sinus could have been transmitted through the attenuated muscle (see pathological report of Case 8 in Appendix II). In my earlier investigations it seemed to me that the auricle must have been paralysed during life, particularly as in these cases no evidence was afforded of the auricular contraction.

§ 156. **Manner in which the nodal rhythm leads to heart failure.** —McWilliam[129] has demonstrated experimentally that when a heart is made to reverse its contraction, the ventricle contracting first, there is at once a great embarrassment of the circulation. It is readily intelligible that the heart being adapted to perform its work most efficiently in such

a manner that the auricles contract and then the ventricles, reversal of this sequence must be against efficiency. When, therefore, the ventricle starts the contraction the heart is at once embarrassed in its work. This embarrassment is made evident by a diminution in the reserve force, and the symptoms produced are simply those that imply an exhaustion of this reserve force.

The degree to which the heart failure may proceed depends on the integrity of the heart muscle. If this be good, then it is able to cope with the embarrassment induced by the abnormal rhythm, and very little change may take place. If it be much degenerated, and particularly if the inception of the rhythm is accompanied by excessive rapidity and dilatation of the heart, then the condition becomes speedily very grave.

§ 157. **Classification.**—In the analysis of over 600 cases where this abnormal rhythm has occurred, I have sought for the facts that would be of use in treatment. With our imperfect knowledge of the cause of this rhythm, it is not yet possible to give a scientific analysis, and I therefore present the matter in the following classification, which will be found to be one readily applied and very useful in practice. As the manner in which the heart carries on its work after the inception of the nodal rhythm depends a great deal upon the rate of its contraction, the cases can be classified as follows :—

(*a*) Where the rate is not markedly increased.

(*b*) Where the rate is greatly increased.

(*c*) Where the nodal rhythm is transient and recurrent (one form of paroxysmal tachycardia).

(*d*) Where the rate is markedly slower than normal (nodal bradycardia, see p. 337).

§ 158. **Cases in which the rate is not markedly increased.** *Symptoms.*—In a great many people this continued irregular action of the heart is found with the rate about normal or very slightly increased (under 90). The change in the heart's action may take place so quietly that the patient may be unconscious of it, and continue at his work, which may be laborious. Usually there is a slight limitation of the field of cardiac response, and many go about quietly for years with little further trouble, even though the legs may become a little swollen. Sooner or later this tendency to oedema increases, yielding at first to treatment but tending to recur, until finally extreme heart failure sets in. This form in rheumatic hearts may occur in early life ; in cardio-sclerosis, rarely before fifty, becoming more frequent with increasing years.

Prognosis.—Many people may show this characteristic irregularity

and be engaged in arduous labour for many years. In such cases if it occurs before fifty years of age, there is usually a past history of rheumatic fever. When it occurs in later life, the extent to which the field of response becomes limited is the best guide. If the field be fairly good patients may go on quietly for many years, some living well over seventy.

Treatment.—Unless there is a distinct evidence of heart failure, no treatment is necessary. If the heart-rate begins to increase in frequency and dropsy sets in, then the treatment should follow the lines laid down in the chapters on treatment.

§ 159. **Where the rate is greatly increased.** *Symptoms.*—The sensation felt by the patient when this rhythm is first started may be so slight as to pass unnoticed. Usually he is conscious of a curious fluttering sensation inside the left chest. This sensation is very characteristic and almost pathognomonic. It may be described variously according to the literary gift of the sufferer, but the essential feature is the soft and gentle movements, not rhythmical, but varying softly in intensity, in striking contrast to the sensations that may arise from stimulation of the heart during a normal rhythm, as in palpitation. Frequently this sensation is so disquieting that the patient rests, or walks about cautiously and quietly. About the same time the patient remarks a distinct limitation in the field of cardiac response, the exertion that he was wont to undertake with comfort now inducing breathlessness.

The associated heart failure may be so extreme that in a few days, or even in a few hours, evidences of imminent peril are shown. The patient has to keep in bed, the dyspnoea being so great that he cannot lie on his back, but must be propped up. If the patient survives, oedema of the legs quickly supervenes, the lips become livid and the face swollen. The pulse is small, rapid, and usually irregular, the beats varying remarkably in strength. The character of the heart's action at this stage can be realized from such tracings as Figs. 107, p. 167, and 110, p. 168. The veins of the neck are often full and pulsate with great rapidity. The heart dilates in a few hours, sometimes extending two inches in the transverse direction. The sounds alter, becoming short and sharp, and if the heart's action is rapid, often no murmur may be detected. If there has previously been a pre-systolic mitral murmur, it disappears, and if the heart's action is fairly slow a diastolic mitral murmur may be detected, but never a pre-systolic. The liver becomes enlarged, and may be found pulsating two or three inches below the ribs. The tissues covering the heart and the liver often become extremely tender on pressure.

If the heart does not slow down after this sudden inception of the

nodal rhythm, the patient drifts on to death, and many cases of rapid and fatal breakdown, particularly in old people, are directly due to the inception by the heart of this rhythm (Cases 11, 12, and 15, Appendix II). Usually the heart does slow down and the circulation improves, and the patient may so far recover as to be able to go about for years with more or less comfort, but is always liable to relapses more or less severe. One often comes across people who have had a continual irregularity of the heart's action for many years, and they have learned to treat themselves with remarkable success, and some of the more intelligent can give the physician very important information as to the relative value of many heart drugs, as digitalis and strophanthus.

Prognosis.—The prognosis depends on how the heart behaves, and how well it maintains the circulation under the new rhythm. If the patient feels little or no distress then he may be assured of many years of fairly comfortable life. If there is some distress in breathing, or a tendency to 'bronchitis' and swelling of the legs, he may still live for many years, his life chequered by attacks of heart failure more or less severe. If the condition be extreme with rapid heart and much dropsy and dyspnoea, the prognosis depends on how he reacts to treatment. The young, or those with a rheumatic history, usually rally from their breakdown, and may be for many years free from attacks. The elderly also rally from their first attacks, and may live for years a somewhat crippled existence. But, as I have already indicated, the persistence of a rapid heart, and evidence of failure of the heart to maintain the circulation, in spite of treatment, are evidences of a very serious kind.

Further evidence will be found in estimating the size of the heart, as dilatation in these cases is always an indication of the failure of tone, and with the permanent failure of tone the work of the heart is further grievously embarrassed (Chapter XXIII).

Treatment.—It should be borne in mind that the condition is the outcome of a series of changes that have been going on for years, and that it is foolish to suppose that by any means we possess we are able to remove the diseased processes on which this arrhythmia depends.

Attempts, therefore, to 'cure' the diseased condition are futile. What we have to do is, not to waste time in the pursuit of a hopeless quest, but to make the best of an irremediable condition, and in recognizing this we are at once placed in a position to render real help.

When the change in rhythm is accompanied by little distress, no treatment is necessary. When, however, there is evidence of distress, treatment should follow in the main the principles laid down in the chapters on

treatment. If there be extreme difficulty in breathing and great restlessness, it is of the first importance that rest should be secured, and that the patient should sleep. For this purpose sedatives are necessary, and they should be given on the lines laid down elsewhere.

A curious and instructive reaction to digitalis will be found in these cases. Most of those whose arrhythmia is of rheumatic origin show a remarkable sensitiveness to digitalis, the heart sometimes responding to a very few doses, and at the same time becoming much slower (see § 251 and Appendix VI). On the other hand, in many cases in which the arrhythmia arises secondarily to cardio-sclerosis, the digitalis has no effect upon the heart.

§ 160. Where the nodal rhythm is transient and recurrent (paroxysmal tachycardia).—In this class the heart suddenly takes on the nodal rhythm, and this may last for a few beats, or it may go on for minutes, hours, days, or weeks. The rate is usually greatly increased (200 per minute and over), and is then recognized as 'paroxysmal tachycardia'. Sometimes the rate is not markedly increased, and in rare cases it may be slower than normal (Case 17, Appendix IV). An individual may have but one attack, or the attacks may come on at frequent intervals in the course of ten or twenty years, or they may be of great frequency, occurring every few weeks or days; or there may be several attacks in one day. After one or two attacks the heart may settle down permanently with the nodal rhythm.

The term 'paroxysmal tachycardia' has been used to describe several conditions that present a superficial resemblance, but are fundamentally different in origin. While the nature of the rapid action was unknown the term served a useful purpose; now it should not be used without a clear definition of what is meant. Though many cases with a normal rhythm have been included under this term, the most striking instances described as paroxysmal tachycardia in literature are undoubtedly due to the inception of an abnormal rhythm; so far I have been able to make out two forms of paroxysmal tachycardia, the transient nodal rhythm referred to here, and an auricular form described on p. 334.

Symptoms.—In extreme cases the sensation of the patient and the associated heart failure are exactly those described in § 159. The rhythm in such cases may be quite regular, and the rate may be over 200 beats per minute. The changes described in § 159 may appear in a few hours. With the sudden reversion of the rhythm to the normal, the change in the patient's condition is even more remarkable than the rapid onset of the symptoms of heart failure. At once the patient heaves a sigh of relief, and in a very short time, within half an hour, all abnormal signs in the lips, face, and

enlarged liver disappear, while in a few hours the heart may be found beating within its normal limits.

In others, the paroxysmal tachycardia may occur for a day or two, with no marked change in the size of the heart. In these, the patient is generally conscious of the heart's abnormal action, and instinctively avoids active exertion, either keeping in bed or resting in a chair, or walking about very quietly. The attack usually lasts for a few hours, but may occasionally last for many hours, or even one or two days. At the end of this time no increase in the size of the heart is detectable, and there is no sign of dropsy or enlarged liver. This condition may be found at all ages over ten years (Appendix II).

Prognosis.—The prognosis in these cases is one of considerable difficulty. The symptoms during an attack may be so alarming that the inexperienced are apt to look upon the patient as hopelessly stricken. I remember being called one night to a woman eighty years of age, I found her heart extremely rapid and irregular, her face swollen and livid, and she was gasping for breath. I told the friends the end was approaching, and, calling to see her next morning, I found her walking out in the street. After that she had several attacks and finally died during one. Other patients I have watched for years, when the symptoms have not been so extreme, sometimes only giving rise to a slight uneasiness in the chest. In these the heart has not dilated, though the rate may have reached nearly 200 per minute. Others have had but one attack, and I have watched some of these for nearly twenty years and they have had no other.

In some the change from being transient becomes permanent, and here lies the danger. If the extreme form of heart failure persists and the dilatation cannot be reduced, then the patient drifts to death (Cases 10, 11, 12, and 15, Appendix II).

The prognosis also depends on the degree of dilatation. If the heart does not increase in size and the attacks are transient, then on the whole the prognosis is good, though in many cases the patient's life is greatly crippled because of the fact that the attacks recur in spite of treatment. When there is dilatation with the accompanying symptoms of dropsy and enlarged liver, then the outlook is bad.

Treatment.—Absolute quiet is needed during the attack—a suggestion scarcely necessary, for the patient usually seeks rest, though he may move about quietly with little distress. A great many suggestions as to the arrest of these attacks may be found scattered through medical literature. As the attacks are commonly but transient, some remedy employed opportunely seems to the inexperienced onlooker to restore the heart to its normal

action. Many patients themselves have a knack of doing something that seems to change the rhythm. Sometimes attacks can be stopped by the patient simply taking a series of deep breaths, by slapping the chest, by the sudden application of cold water to the chest. Drugs of the most diverse character have been cited as active agents in stopping attacks, such as nitro-glycerine and adrenalin. In my early days I, too, thought I knew how to stop attacks, but more extended experience has shown me that when they stopped it was from some cause unknown to me, and which was independent of any means I employed.

When signs of heart failure appear the treatment should follow the lines laid down in the chapters on treatment.

CHAPTER XXI

Affections of the Conducting Functions of the Primitive Cardiac Tissue (Heart-Block, Adams-Stokes Disease, Ventricular Rhythm)

§ 161. **Definition.**—In the last two chapters the irregular action of the heart was ascribed to the contraction of the heart starting lower down in the primitive tissue than the sinus. In this chapter another important function of this tissue is considered, namely, the conduction of the stimulus from auricle to ventricle. The stimulus for contraction reaches the ventricle from the auricle by passing along the bridge of primitive tissue that connects the auricle and ventricle (Fig. 2, p. 15). This bridge may be so affected that (1) the stimulus is delayed ; (2) the stimulus is at times prevented from crossing over ; (3) the stimulus may be completely blocked beyond the a.-v. node, and the ventricle then contracts in response to a stimulus that arises in the uninjured remains of the a.-v. bundle (heart-block, ventricular rhythm).

§ 162. **Methods of recognizing depression of conductivity.**—Apart from suitable tracings, the clinical evidence is limited to the recognition of the fact that there is a slow radial pulse, or, better, a slow ventricular rate, while the veins in the neck pulsate more frequently, owing to the normal rate of the auricular contractions being maintained. If the ventricular contractions are over thirty-six per minute, these may have a distinct relationship to the auricular waves in the jugular pulse, the ventricle

responding to every second, third, or fourth auricular contraction. When the ventricular contractions are about thirty or under, they are probably independent of the auricle, and auricle and ventricle beat at independent rates and in response to independent stimuli.

When there is a mere delay in the response of the ventricle to the stimulus from the auricle, the evidence is very scanty, apart from graphic records. I have, however, in cases of mitral stenosis been able to recognize this delay by a slight separation of the presystolic murmur from the first sound of the heart (represented diagrammatically by the shading in Figs. 112, p. 177; 272, p. 365, and 265, Plate V). Occasionally 'missed beats' or pulse intermissions are due to the auricular stimulus failing to provoke a ventricular contraction, and a strong suspicion of the nature of this irregularity may be aroused by observing that during the pause the ventricular sounds are absent; this distinguishes it from most cases of extra-systole, in which, as has already been described, there are usually heard two short, sharp sounds, the result of the weak, short, premature contraction of the ventricle. As, however, extra-systoles may occur with no audible sound, this distinction is not reliable. In tracings of the radial pulse alone, Wenckebach [228] was able to recognize the nature of the irregularity by an ingenious method of measurement. No difficulty is presented, as a general rule, when tracings of the radial pulse or apex beat are taken at the same time as tracings of the jugular pulse. The slighter forms of this affection are recognized by the delay that occurs between the auricular and ventricular systoles. In the jugular tracings, as has already been shown, there is usually present a wave due to the auricular systole, (*a*) followed at a short interval by the carotid wave (*c*). This interval between *a* and *c* is of great value in estimating the condition of the conductivity in the primitive tissue.

§ 163. **The intersystolic period (*a–c* interval).**—This interval is occupied by three events, namely, (1) the systole of the auricle; (2) the transmission of the stimulus from auricle to ventricle; (3) a minute portion of time during which the ventricular pressure is rising before opening the semilunar valves (*Anspannungszeit*, or presphygmic interval). As 3 is practically constant, it may for the purpose of this inquiry be ignored, and assuming that the stimulus for contraction starts on its way to the ventricle at the beginning of auricular systole, any variation in the length of the *a–c* interval is due to the variation of the rate of stimulus conduction.

In normal hearts I have found that the *a–c* interval is fairly constant, lasting usually one-fifth of a second (as in Fig. 44). It is a little shorter in frequent action of the heart.

§ 164. **Depression of conductivity without arrhythmia.**—While the *a–c* interval may be considered normal when it does not exceed one-fifth of a second, considerable increase of this interval may take place with no interference with the rhythm of the heart.

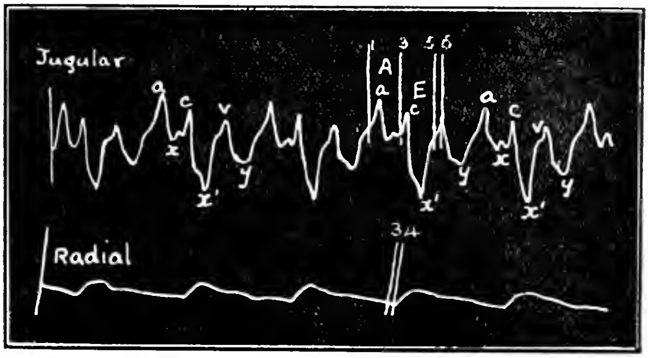

FIG. 111. Shows a great increase in the *a–c* interval (space *A*) due to a delay in the stimulus passing from auricle to ventricle. (Case 17, Appendix IV, taken 1892.)

In Fig. 111 there is a tracing of the jugular pulse taken at the same time as the radial pulse. The radial shows a perfectly regular rhythm, while the neck tracing shows a great increase in the *a–c* interval (space *A*). The heart may continue to beat perfectly regularly for years with the

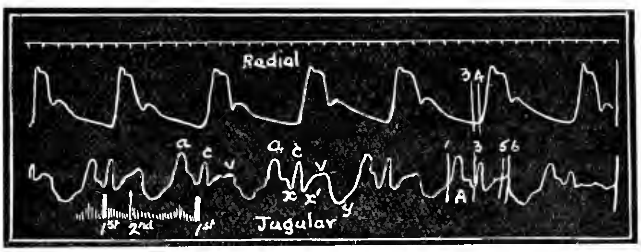

FIG. 112. Shows an increased *a–c* interval, nearly two-fifths of a second in duration (space *A*). The shading shows the position of the murmurs—a short murmur due to the auricular systole, the loudest part of which is separated from the first sound by a brief interval, a murmur following the first sound, and another following the second sound running up to the presystolic (or auricular systolic) murmur. (Case 17, Appendix IV, taken 1903.)

conductivity affected to this extent. Thus, Fig. 112 was taken in 1903 from the same patient from whom Fig. 111 was taken in 1892, and the jugular tracing shows a like increase of the *a–c* interval (space *A*). Except for a short period in 1898, this patient's pulse was quite regular up to 1904.

§ 165. **Influence of rest upon conductivity.**—With each contraction

all the functions of the muscle-fibres are for the time being abolished, to be gradually restored during diastole. Normally, their recovery is simultaneous, so that when the rhythmical stimulus arises at the auricle or great veins it passes over the whole heart, and the muscular fibres respond at a uniform time. When one of these functions is depressed, its recovery does not take place as speedily as the others, therefore the relationship of the action of these functions is not uniform, and an interference with the regular sequence of events results. Certain variations in the rate of conduction can frequently be detected, not only when conductivity is depressed, but also when it is presumably normal, such variations apparently depending on the time required for recovery after the previous exhaustion. One has little difficulty in illustrating this in a variety of ways in certain forms of heart irregularity. In a patient aged 24, suffering from mitral disease,

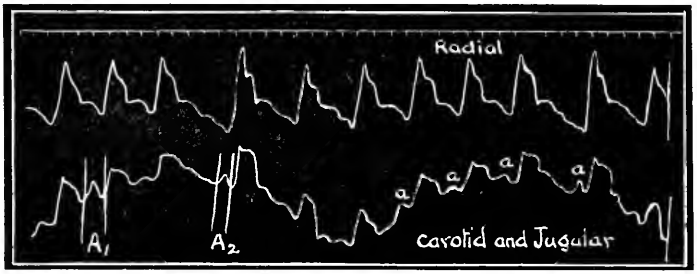

Fig. 113. Shows the influence of rest when the conductivity of the a.-v. fibres is depressed. The radial pulse shows a respiratory irregularity. The carotid and jugular tracings show a shortening of the *a-c* interval (space A_2) after the long diastolic periods, and a lengthening (space A_1) after the short diastolic period of the cardiac cycle.

and pregnant with her first child, I had noted an increase in the *a–c* interval for some years. During labour it frequently happens that the pulse becomes irregular, the irregularity often being of a respiratory type (sinus irregularity). The radial tracing in Fig. 113 shows such an irregularity. The tracing of the carotid and jugular shows a rise and a fall due to the movements of respiration—the rise in the tracing corresponding with expiration, and the fall with inspiration. It will be noted that the irregularity is due to a lengthening of the diastole, and that the *a–c* interval during the short pulse period (space A_1) is nearly double the duration of the *a–c* interval during the long pulse period (space A_2). The reason for this variation in the *a–c* interval is that the conductivity is continually depressed, but when the heart beats at a slower rate the a.-v. fibres have time to recover during the longer diastolic pause, so that the stimulus is conveyed from auricle to ventricle at about the normal rate (A_2). Here the arrhythmia is not due to the depressed conductivity, but evidently originates in the sinus, for the auricular wave,

a, appears at irregular intervals. The modifying effect on the ventricular rhythm of the variable condition of the conductivity can be illustrated in a variety of ways. If we examine minutely the incidents where an occasional premature or extra systole occurs, sometimes very curious variations in conductivity can be detected (as in Fig. 106, Plate II; also Figs. 274, and 265 Plate V).

It may seem an unnecessary refinement to dwell upon such minute changes, but I trust to show later that the recognition of these facts affords the evidence for determining with certainty some changes of the utmost significance in a large class of cases of heart disease.

§ 166. **Arrhythmia due to depression of conductivity.**—Slight arrhythmia may be due to variations of conductivity. Thus the short space

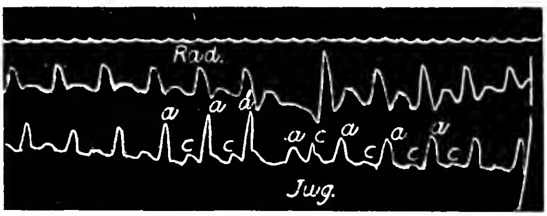

Fig. 114. Shows a gradual lengthening of the *a–c* interval till the stimulus from the auricle at *a′* reaches the a.-v. bundle before the latter has recovered from the previous stimulation, and finds it 'refractory'. The ventricle, therefore, does not respond to this stimulus, but remains quiescent till the next physiological stimulus comes from the auricle, and the conductivity being restored the ventricular beat (*c*) follows the auricular wave at a shorter interval. Note the increasing size of the *a* wave before the intermission. This is due to the auricular systole falling at the same time as the preceding ventricular systole, so that the auricular contents cannot be sent into the ventricle, but a bigger wave is sent back into the veins.

o in the radial in Fig. 98 is seen to be due to an increase of the preceding *a–c* interval (space A_1), that is, to a delay in the ventricular systole.

Arrhythmia of a more marked character arises when the conductivity is so grievously depressed that the stimulus occasionally or frequently fails to cross the auriculo-ventricular junction. How this occurs is well seen in Fig. 114. There is a constant delay in the conduction of the stimulus here, the *a–c* interval being unduly prolonged. Before the intermission there is a slight but gradual increase of the *a–c* interval. After the auricular wave *a′* there is no carotid wave (*c*) nor pulse beat in the radial. The reason for this is manifestly that the auricular systole *a′* occurred so soon after the previous ventricular systole (as evidenced by the carotid wave *c* immediately before *a′*) that there was not sufficient time for the recovery of the function

of stimulus conduction in the a.-v. bundle, and hence the stimulus failed to reach the ventricle, and a beat dropped out. By this means a longer rest was procured for these fibres, and when the next stimulus comes down from

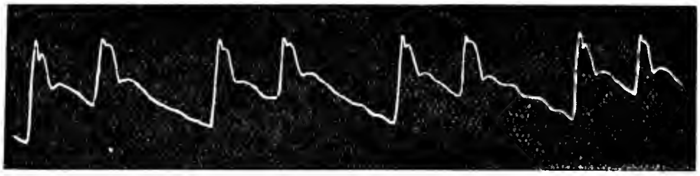

Fig. 115. Regularly intermitting pulse due to depression of conductivity. (Case 17, Appendix IV, 1898.)

the auricle the longer rest has so restored the function of conductivity that the *a–c* interval following the pause is shorter than the average. This dropping out of ventricular beats may occur at regular intervals. Fig. 115 was taken in 1898 from the patient from whom Figs. 111 and 112 were taken.

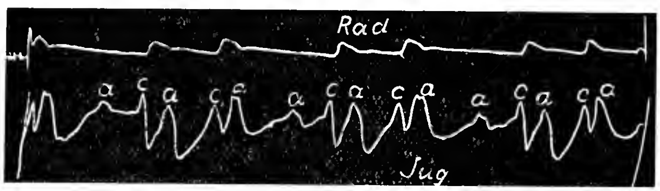

Fig. 116. Taken at the same visit as Fig. 115, and shows the venous pulse during the arrhythmia. The wave *a* is quite regular in its appearance. For the interpretation of this tracing see diagram Fig. 117.

For many years tracings from this patient showed constant depression of the conductivity. For some reason, in 1898 the conductivity became further depressed, so that at regular intervals a ventricular beat dropped out (Fig. 115). The true nature of this arrhythmia is shown in the jugular tracing

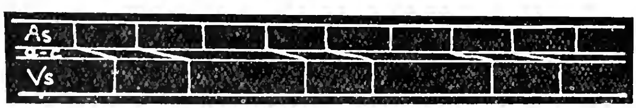

Fig. 117. Diagram constructed to show that the irregularity in Figs. 115 and 116 is due to a blocking of the conductivity at the fibres joining auricle and ventricle. Note the increased length of the *a–c* interval before the pause in the *Vs*.

(Fig. 116), where the auricle is shown to contract regularly (wave *a*), while the ventricle fails to respond to every third auricular systole. To demonstrate this more clearly, I reconstruct Fig. 116 in the form of a diagram (Fig. 117). The downstrokes in the upper division represent the auricular

systole (*As*), those in the lowest the ventricular systoles (*Vs*), and correspond with the radial and carotid pulses in Figs. 115 and 116. The slanting lines represent the *a–c* interval. It will be seen that a ventricular beat drops out regularly after every third auricular systole.

§ 167. **Missed beats due to depression of conductivity.**—Gaskell[123] states that on applying a screw clamp around the auriculo-ventricular

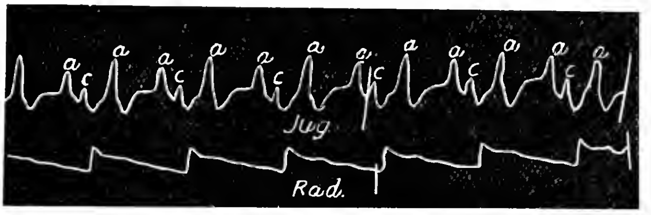

Fig. 118. The ventricle only responds to every alternate auricular systole—ventricular rate 48, auricular rate 96.

groove of a frog's heart, ' according to the tightness of the clamp the ventricle can be made to beat synchronously with the auricles, to respond to every second contraction of the auricle, to respond to every third, fourth, or other contraction, or to remain quiescent.' Hering[295] and Erlanger[272] have recently produced the same changes in the mammalian heart. All these varying results can be demonstrated to occur in the human heart.

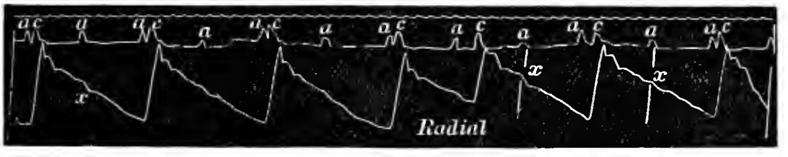

Fig. 119. Is from a slow, irregular pulse after influenza, and the jugular tracing shows that the slow pulse is due to the ventricle failing to respond to the stimulus from the auricle. Note that after the short pulse period in the radial the *a–c* interval is much longer than at the other periods. This is because the fibres have had a short rest, and the conductivity has in consequence not been completely restored. Note also at *x* a slight depression in the radial tracing due to the systole of the left auricle affecting the arterial column (see Fig. 120).

In Fig. 114 the ventricular systole is seen to drop out at rare intervals; in Fig. 116 the ventricular systole drops out after every third auricular systole (3:2 rhythm); in Fig. 118, after every second (2:1 rhythm). In Fig. 119 the ventricular systole usually drops out after every second auricular systole; but there is one short pulse period, and the *a–c* interval here is much longer than the *a–c* period after the longer periods, the lengthening being an evidence that the conducting power of the auriculo-ventricular fibres has not had time to recover as effectually as after the longer pulse periods

(see diagram, Fig. 120). In Fig. 121 there are three auricular contractions to one ventricular (3:1 rhythm), except during the last arterial pulse-period, when there are but two auricular waves, and after the second of these the *a–c* interval is longer than the other *a–c* intervals in this tracing.

In tracings published by G. Gibson [8], Hay [288], and Beards [259], there are four auricular beats to one ventricular. This blocking may be so extreme that the auricle may beat ten or twelve times and the ventricle stand still.

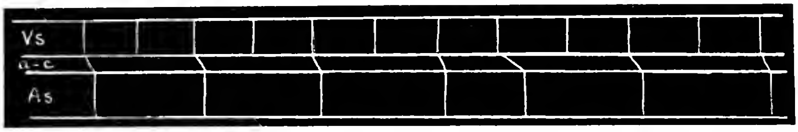

Fig. 120. Diagram of Fig. 119, showing the blocking of the stimulus after every second auricular systole, except in one instance when it gets through, with a lengthened *a–c* interval.

§ 168. Independent ventricular rhythm due to heart-block.—In the tracings I have given so far, it could be shown that when the ventricle does contract it is in response to a stimulus from the auricle. When a ligature is applied in the auriculo-ventricular groove of the frog's heart, so that the stimulus can be conveyed no longer from auricle to ventricle, the latter beats, after a time, with a rhythm different from, and independent of, that of the auricle (complete heart-block). Wooldridge and Tigerstedt produced complete independence of the auricular and ventricular rhythms by physiologically separating the auricles from the ventricles, while a similar result has been attained by His, jr., Hering and Erlanger through compression of the a.-v. bundle. Erlanger [272] has produced complete heart-block experimentally in dogs, and the dogs have lived for many months, with symptoms identical with those found in human subjects suffering from heart-block. This independent rhythm can be demonstrated as the cause of certain forms of a slow pulse-rate in the human subject. Fig. 122 is a tracing of the radial taken at the same time as the pulsation in the neck

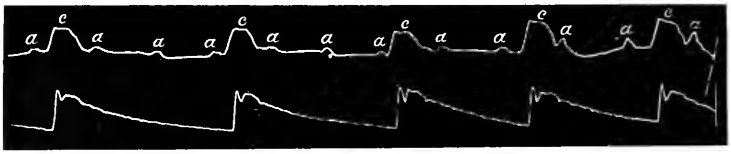

Fig. 121. Here the tracing from the neck shows sometimes three auricular waves (*a*) to one carotid wave (*c*). In the last two periods there are but two auricular waves, and the *a–c* interval is longer than in the periods when there are three auricular waves, because in the former case the conductivity has not had so long a time to be restored.

due to the jugular and carotid. The small waves, *a*, are due to the right auricle, and there are two of these auricular waves to one carotid or radial pulse beat. But when one carefully analyses their relationship, it is found that the two auricular periods are less than one ventricular period, so that the

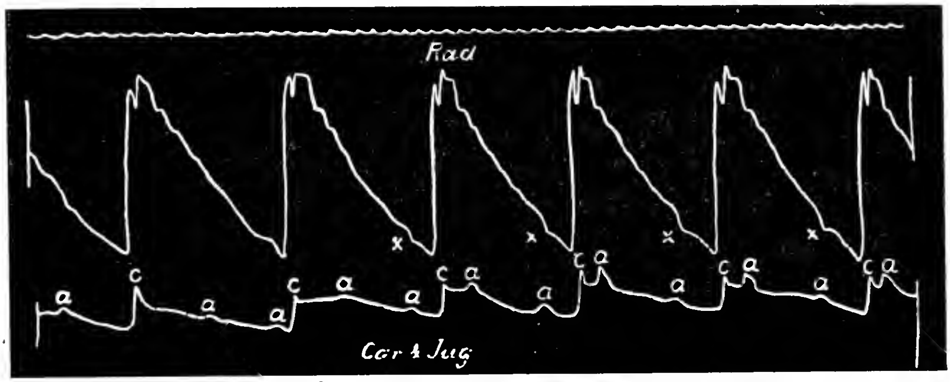

Fig. 122. Shows complete heart-block, the auricle pursuing one rhythm, and the ventricle another and slower rhythm.

relationship of the auricular systole to the ventricular systole is a constantly varying one, sometimes at a distance, and then gradually approaching till they are synchronous. Whatever relationship the *a* has to *c*, no variation takes place in the rate. The same thing is shown in Fig. 123, where the apex

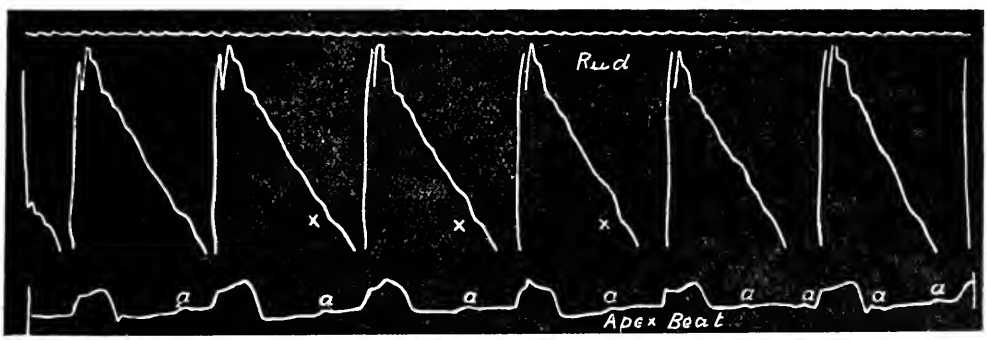

Fig. 123. From the same case as Fig. 122. Shows the influence of the left auricle on the apex tracing (wave *a*). In the radial tracing the dips (×) due to left auricle are found to occur immediately after the auricular waves in the apex tracing.

beat is recorded at the same time as the radial. Here there is a small wave, *a*, due to the systole of the left auricle, and the same maintenance of independent rhythms is manifest here. One can infer with certainty that the auricle contracted at the same time as the ventricular systole during the first four

or five of the ventricular apex beats, but that the evidence of this simultaneous contraction is obscured by the large ventricular wave, for we can see in Fig. 122 how the wave *a* occurred at the same time as the carotid wave. I have taken a large number of tracings from this patient at various times and under various circumstances, and I have invariably found the independence of rhythm between the auricle and ventricle.

§ 169. **Effect of the auricular contraction on the radial pulse.** —The auricular systoles can sometimes be recognized in another way in tracings of the radial pulse, where, during a long ventricular pause, a series of notches can be seen occurring at regular intervals on the descending line of the radial tracing, as in Fig. 124. If a jugular tracing be taken at the same time, there will be found auricular beats occurring exactly at the same time as these notches, as is shown in Figs. 119, 122, and 123. From this I suggest that those notches in Fig. 124 are due to the movement of the

FIG. 124. From Webster's case of heart-block, showing in the falling line of the radial tracing a series of interruptions (*a*) due to the left auricle.

left auricle, the systole of the left auricle pressing against the aortic valves temporarily affecting the arterial column. The sudden cessation of this pressure causes a slight but abrupt fall in the aortic pressure, giving rise to the notches in the radial tracing.

§ 170. **Etiology.**—Except in cases of complete heart-block, the anatomical changes have not been fully worked out. In the majority of the cases of heart-block examined post mortem, damage of the a.-v. bundle at or beyond the a.-v. node has been found, due to acute inflammatory changes or to sclerotic changes, or to the presence of gummata. In the milder cases one can only infer the cause. From Gaskell's experiments we know that if the bridge of muscle connecting the auricle and ventricle be narrowed, the stimulus takes longer in passing. We know also that in rheumatic affections of the heart numerous deposits of cells occur in the muscle substance. As it is in rheumatic heart cases and cardio-sclerosis that I have found most of the milder forms of depressed conductivity, I infer that when such deposits occur, or when a slight cicatrization takes place, the bundle is injured, and so the function of conductivity is impaired. In other acute affections of the heart we get evidence of the implication of this

bundle in the diseased process (see Chapter XXIV). Little is known as regards complete heart-block as an acute condition, but I refer to its possibility in febrile affections of the heart, and one case has been recorded by Jellinek and Cooper[301] with confirmatory post-mortem details. In cardio-sclerosis we may observe symptoms of impaired conductivity and even heart-block in an individual who has shown extra-systoles and the nodal rhythm.

The sclerotic changes in the heart muscle associated with arterio-sclerosis of the coronary arteries appear likely to be the most common cause of heart-block. If one looks at Fig. 2, it will be seen how the a.-v. bundle passes through the central fibrous body of the heart. This body is composed of fibrous tissue, and is the fixed point in which are inserted many of the muscular fibres of the ventricle, and from this place sclerotic changes are apt to spread. Further, Keith's researches into the hearts I have sent him have shown that the sclerotic process is apt to extend from the base of the mitral valve into the central fibrous body, and to involve the bundle. I have suggested this as a probable cause for the production of extra-systoles and the nodal rhythm, and, in certain advanced cases, of heart-block.

Several cases have been reported where the damage to the bundle was due to gummata, and the recovery of other cases under anti-syphilitic treatment indicate that syphilis may be an agent in the production of complete heart-block. Bramwell[262] has recently reported a case of complete heart-block in a rheumatic heart, and I have found that slight impairment of the bundle in rheumatic hearts, especially with mitral stenosis, is not infrequent.

In complete heart-block the a.-v. node may be either destroyed or separated from the remainder of the a.-v. bundle. The remains of the bundle in the ventricle beyond the a.-v. node have been found perfectly normal in a case described by Keith in which there was a history of complete heart-block for eighteen years. Seeing that the bundle was healthy, it must have had some function during life, and as it did not convey stimuli from auricle to ventricle, it must have had some other function, which I suggest was stimulus production, and the maintenance of the slow ventricular rate.

There is a great defect in our knowledge of the condition leading up to heart-block. Before the slow ventricular rhythm becomes permanent, in many people there are periods in which, though the rhythm of the heart is normal, there is frequently an increased *a–c* interval ; and at times ventricular systoles drop out at intervals more or less regular and frequent. I am convinced that in some cases there is a connexion between the conditions producing the nodal rhythm and heart-block. I have reasoned that most cases of nodal rhythm are due to degeneration or irritation of the a.-v. node,

while a few may be due to a break between the sinus remains and the a.-v. node on account of the atrophy of the auricular wall from long-continued distension. The mischief causing the nodal rhythm and the independent ventricular rhythm of heart-block is thus seen to be very near. I cite fully in the Appendix (IV) cases of nodal bradycardia having some affinity to heart-block. In one there was for years an increased *a–c* interval, and at one period a mild heart-block, and finally a nodal rhythm of a very slow character (Case 17, Appendix IV).

The possibility of vagal stimulation taking part in the production of heart-block must be borne in mind. Stimulation of the vagus can produce a similar action of the heart, as Chauveau [265] pointed out. Roy and Adami [178] give excellent tracings showing complete heart-block due to direct stimulation of the vagus and to the administration of muscarin. I give tracings where a mild form of heart-block was produced by a reflex stimulation of the vagus by swallowing (Case 27, Appendix VI). Digitalis may also produce it. But in these cases there is evidence of a defect of the a.-v. bundle—in the permanent delay of conduction. In Webster's [326] case the arrest of the ventricle arose in two ways, as Wenckebach's [328] masterly analysis brings out, and one of these ways points to vagus stimulation.

§ 171. Significance of the milder forms of depression of conductivity.—It must not be thought that the foregoing data are merely of academic interest. Their recognition and appreciation clear up many obscurities surrounding heart affections, and are of practical importance in treatment. As will be shown, in acute affections of the heart the presence of the irregularity due to this cause indicates that the muscle is being invaded by the disease. When it occurs without any irregularity, the increased *a–c* interval is of importance in the administration of such drugs as digitalis. I have rarely failed in such cases in increasing the *a–c* interval and causing the dropping out of ventricular systoles by the administration of digitalis, and the recognition of this form of irregularity produced by digitalis is of importance, for digitalis should never be pushed further. One reads of accounts of sudden death during or after the digitalis had slowed the pulse, and it has seemed to me that the immediate cause might be the production of severe heart-block and consequent syncope. I have a few times seen in consultation patients whose hearts were greatly slowed by excessive doses of digitalis, but at that time I had not acquired the means of recognizing the cause. I always make it a rule to stop digitalis as soon as I find the pulse dropping a beat (see § 252). The recognition of the cause of the intermission, as in Fig. 115, should always be an indication against the use of digitalis.

§ 172. **Symptoms associated with heart-block.**—Apart from the characteristic irregularity, and the slow ventricular rhythm with its associated syncopal attacks, there are no characteristics. Patients with a pulse-rate of thirty to forty beats per minute may go about their affairs, but quietly—the field of response being distinctly limited. The manner of limitation is varied: in some it is a sense of weakness, in others exertion induces attacks of dyspnoea—sometimes extremely violent on very slight exertion. The picture given by the late Sir W. T. Gairdner, a sufferer from heart-block, of his own experiences, is very characteristic of some cases. 'I am wonderfully free'—he wrote me four years after the slow rhythm began, and two years before his death at the age of eighty-two, his pulse-rate then being thirty per minute—'from all the symptoms that usually go along with organic heart-disease. My sleep is almost always undisturbed, and I get abundance of it both by day and night, nor is there the slightest trace of angina pectoris, severe dyspnoea, dropsy, or any of the usual accidents of prolonged cardiac disease.' He wrote later: 'Although a little uncertain in my gait, I can go from one room to another or even up a simple stair, taking plenty of time and assisted by the railing; but for the last two years at least, if not more, my position has been with few exceptions recumbent, or at most sitting, and repeated attempts have shown me that it is practically impossible to cross the street or to go into the garden opposite the house except in a wheeled armchair; and along with this there is a feeling of perpetual weariness which never leaves me even after the soundest sleep, and which is not explained by any pain or suffering, though in itself it often tends to fits of yawning and even exclamations which would sound to others as if I was suffering inwardly' (see also Gibson and Ritchie's [232] description of this case).

The syncopal and epileptiform attacks due to cerebral anaemia induced by the slow action or temporary stoppage of the ventricles (Adams-Stokes syndrome) have been described on p. 24. This tendency to infrequent action may appear in affections of the bundle at two stages, namely, before the permanent establishment of the independent ventricular rhythm and after its establishment. While there are intermittent periods of temporary heart-block, there is a certain liability to these syncopal attacks, and the following appears to me to be the reason. After a Stannius' ligature is applied between auricle and ventricle, the ventricle stands still for a longer or shorter period before it starts on its own rhythm. In certain cases of heart-block the stimulus to contraction passing from auricle to ventricle may suddenly stop, and the ventricle pause for a brief period before it starts its own contraction. It is during this period that the syncopal attacks occur. Thus,

Sir W. T. Gairdner noticed that his attacks always came on with the sudden dropping of his pulse-rate : ‘ These cardiac and cerebral attacks were at one time so frequent that I think from twenty to thirty of them were numbered in twenty-four hours.’ His pulse-rate would be seventy per minute, and after his syncopal attack he invariably found it at or below thirty per minute. When the pulse subsided to a permanent rate of twenty to thirty per minute, the cerebral attacks disappeared. My reading of this description is that while the block was partial the stimulus at times got through from the auricle and the rate of the pulse rose to seventy ; then the stimulus suddenly failed to get through, and the ventricle paused for a brief period as it does on first applying the Stannius' ligature, anaemia of the brain resulted, and the patient fainted. On the ventricle starting its own rhythm the circulation of the brain was restored, and the patient, recovering, found his pulse at the rate of thirty per minute. When, however, the block became permanent, the ventricle went on at its slow, independent rate, with no pauses and therefore with no syncopal attacks. From tracings taken during attacks of syncope, Wenckebach and Gossage have shown that the cerebral anaemia may arise by the ventricle suddenly beating with great rapidity, the individual contractions, however, being so small that the brain is not sufficiently supplied with blood.

Syncopal attacks may appear when the rate is constant at thirty and under. In such cases there is invariably a greater slowing of the heart's action, the rate falling sometimes as low as five beats per minute. This point is well brought out in the tracings from patients during an attack taken by Webster [323], Hay [290], and Barr [257]. The immediate cause of the greater slowing is not understood.

I have only seen a patient recovering from an attack, and I take the following description, which seems fairly representative, from Hay and Moore's [291] description :—

‘ He was sitting up in bed when suddenly another attack seized him ; no sooner had he recovered and spoken a few words, than he was again attacked. He made one or two hurried respirations, his head fell on his breast, and he sank on to his pillow, breathing stertorously ; his cheeks and alae nasi flapped in and out ; he ceased breathing, his eyes closed, but his pupils were widely dilated, and his eyeballs were turned upwards and to the left. His face was ashen grey, the malar flush had vanished, and he had all the appearance of a corpse. In a little, however, the colour gradually returned to his face, he sighed deeply and woke up. He raised himself a little, stared about, and then sank back on the pillows exhausted and sweating. During the seizure the radial pulse could not be felt until just before the return of consciousness.’

In other descriptions epileptiform movements are also included.

From the patient's sensations of an attack, I quote the following letter written by an old friend of mine who suffered from Adams-Stokes syndrome due to nodal bradycardia, the pulse-rate usually being thirty per minute:—

'At your request I try to give you a description of an extraordinary swoon I had whilst suffering under heart trouble. It happened in the middle of the night. I awoke from a quiet sleep feeling a most curious creepy sensation; my functions all seemed to be stopping, and in front of me, about two feet from the floor, appeared a circular light about two inches in diameter, and brilliant beyond anything I had ever seen before. I thought the period of my dissolution had arrived. I was perfectly calm, and I began to reason with myself whether I should waken my wife (I don't know whether I should have had strength; I was turned from her at the time), in which case she would be greatly alarmed, or leave things to take their course. Before my mind was made up what to do, the light began to contract, and when it was reduced to about half its original size suddenly went out, but before entirely losing consciousness I had such a feeling of peace and restfulness as I never experienced before, and had just time to say to myself, "There is no after-life anyway."

'How long I lay in that condition of course I don't know, but when I did come to, I felt it utterly impossible to move: I might have been a leaden image, I felt such a weight. For a long time I persevered in trying to move a limb. At last I got a little life in one of my feet, and then gradually the use of all my limbs.'

Cheyne-Stokes respiration may appear in patients suffering from heart-block, and Gibson and Ritchie [231] record falls of arterial pressure during the apnoeic stage.

A curious feature in cases of heart-block is that circumstances that usually excite the heart to rapid action have little effect upon the independent ventricular rhythm. Causes of excitement, and the administration of alcohol or chloroform, have very slight or no effect on the ventricle, though the auricular contractions may be rendered much more frequent.

§ 173. **Prognosis.**—In the milder cases where there is a delay in the stimulus passing from auricle to ventricle, or where there may be occasionally the characteristic irregularity caused by the dropping out of a ventricular systole, no grave conditions arise. Its recognition, however, warns us that myocardial changes are involved, and the susceptibility of the patient to the digitalis group of remedies should be borne in mind. Patients with mild forms of heart-block, even where the pulse is continuously about thirty, may lead quiet, uneventful lives for ten or twenty years. When there is a tendency to syncopal attacks, then the patient's life is uncertain, as such patients usually die in one of these attacks, often being found dead in bed

or elsewhere. Death may arise from heart failure, preceded by symptoms of oedema of the lungs and dropsy.

§ 174. **Treatment.**—We occasionally meet with cases where, after repeated attacks of syncope and long-continued slow pulse, the conducting power is restored and the rate becomes normal. But considering the pathological change producing these cases, it will be realized how futile it is, except in syphilitic cases, to attempt to give remedies to cure. Notwithstanding this, many writers prescribe drugs of the most diverse character—even digitalis, which is so demonstrably contra-indicated. Fortunately for the patients most of the drugs are innocuous so far as the heart is concerned. The management of these cases should be directed to seeing that the patient leads a life according to his strength, avoiding anything that induces over-exertion, limiting the amount of food to what is necessary for his limited way of living. While the tendency to syncopal attacks is present, extreme care should be taken that he should not expose himself to the attack occurring in dangerous situations. He can move quietly about with such attendance as his means can afford.

In a few syphilitic cases, recoveries have been effected by the energetic use of anti-syphilitic remedies.

CHAPTER XXII

Exhaustion of Contractility

§ 175. Necessity for recognizing exhaustion of contractility.—Seeing that the cause of all forms of heart failure finally resolves itself into the inability of the contractile force to maintain efficiently the circulation, it is necessary to describe in some detail the part played by the failure of contraction alone. While it is true that heart failure may be induced by the disturbance of other functions, and that the symptoms produced by these commonly dominate the situation—excessive rapidity, irregularity, dilatation —there are also signs evoked by the failure of contractility which are often obscured and overlooked in the confusion created by more pronounced symptoms, but which nevertheless can be recognized and apportioned to their appropriate cause. We sometimes see dyspnoea, enlargement of the liver, and dropsy described as the cardinal symptoms of heart disease, but equally serious conditions of heart disease and heart failure may be present with none of these symptoms. With the exception of dyspnoea, the 'cardinal' symptoms are usually associated with dilatation of the heart, i. e. failure of tonicity. Failure of contractility may occur with little or no increase in the size of the heart, without dropsy and with no marked dyspnoea, and this, if properly appreciated, will be found to throw much light on the nature of the heart disease, and to indicate the line of treatment.

§ 176. The function of contractility.—The circulation is carried on by the force derived from this function of the heart muscle. It is from this source that the arterial pressure is maintained. To understand the results of failure or exhaustion of this function, it is necessary to bear in mind certain of its physiological peculiarities. After each contraction of the muscle-fibres the function is so exhausted that no further contraction can occur until a brief period is allowed for recovery. If the stimulus to

contract is applied too soon, a short and feeble contraction results. If a longer period of rest be allowed, the contraction will be longer in duration and more powerful, so that, within certain limits, the longer the rest, the longer and more powerful the subsequent contraction.

Another feature to keep in mind when considering this matter is that, like all the other functions, this possesses in a high degree the peculiar property of 'reserve force'. It is, in a measure, the too easy exhaustion of the reserve force which is the subject of this chapter.

§ 177. **Conditions inducing exhaustion of contractility.**—These may be summed up in the general statement that exhaustion of contractility occurs when the heart muscle has been exposed to a resistance greater than it can overcome without calling upon its reserve force. This may be brought about by the healthy heart muscle having to meet abnormal resistance, or by a weakened muscle opposing a normal resistance. The following description includes the most striking of these conditions.

(*a*) *Increased frequency.*—A certain rate which we recognize as normal is most favourable to the performance of the heart's work, this rate being that which gives time for the functions exhausted by the contraction to recover. An excessive rate prevents the due recovery of the function, and a call is made upon the reserve force, which thus tends to become exhausted.

(*b*) *Dilatation of the heart.*—A certain size of the heart is also necessary for the perfect performance of contraction, and if the chambers be dilated the contractile force is placed at a disadvantage, and exhaustion results.

(*c*) *Obstruction of the heart's work.*—Exhaustion of this function may arise from embarrassment of its work by valvular defects, or by increased peripheral resistance, or by too great calls made upon it by over-exertion.

(*d*) *Imperfect nutrition.*—As each contraction depends on the supply of appropriate nutriment, any interference with this leads to exhaustion. This may arise from deficient supply, on account of the small output of the heart (as in mitral stenosis), or from narrowing of the coronary arteries from disease. It may be due to the blood containing insufficient nutriment of the special kind required, or containing elements that have a deteriorating effect on the muscle.

(*e*) *Degeneration of the muscle-fibres.*—Finally, there may be changes in the heart muscle itself, fibrous or fatty, which may predispose to a depression of contractility by diminishing the number of the fibres, or by impairing the activity of those that are left.

§ 178. **Symptoms.**—The failure of this function may occur while

the other functions of the heart are intact. Thus it may be present with the heart beating at a normal rate, with the ventricular systole following normally on the auricular, and with the heart of a normal size. If we analyse the symptoms present in such cases where the other functions are intact, we can then refer the symptoms with a fair degree of certainty to exhaustion of this function.

It might at first sight be supposed that failure of the force which keeps up the arterial pressure would show itself first of all by a fall in arterial pressure. This, however, is not the case. So readily does the heart respond to the call from the tissues that the arterial pressure with exhausted contractility may be kept up to an extreme height until the moment of death. What happens first is an exhaustion of the reserve force. So great indeed may the exhaustion be, that a patient may be scarcely able to turn over in bed without distress, and yet the arterial pressure may be abnormally high, and the size of the heart be not increased. The frequency of the pulse and respiration may be normal before and after the effort. The classes of patient where the failure of contractility with the integrity of other functions is seen most strikingly are those of rheumatic hearts with valvular and myocardial lesions, and those of cardio-sclerosis. In the former class are the young and middle-aged who have had rheumatic fever some years before, and have gone on doing the work that they were wont to do when their hearts were healthy. Gradual symptoms of exhaustion appear until they become so clamant that exertion has to be restricted. In like manner, those with cardio-sclerosis have continued their wonted exertion until pulled up by some distressing symptoms. Exhausted contractility produces symptoms in a twofold manner, (*a*) by reflexly calling into play the protective phenomena, and (*b*) by certain changes in the action of the heart.

(*a*) *Reflex symptoms.*—Concerning the first of these, they belong to the class described in detail in Chapter VII, the most striking being the attacks of angina pectoris. Though the group of symptoms included in angina pectoris are more commonly found associated with exhausted contractility in cardio-sclerosis, it may also appear in the young with mitral stenosis. The symptom that usually arrests the patient when the reserve contractile force is exhausted is a suffocating feeling coming on during exertion, and referred to the throat and upper part of the chest, or breathlessness, or a sense of exhaustion. Sometimes it is a distressing consciousness of the heart beat (palpitation), often with excessive rapidity of the heart. In females there may be pain complained of in the chest and left arm, and the tissues there may become extremely hyperalgesic.

In those of more advanced years with cardio-sclerosis, the symptoms

may be the same, though, as I have said, angina pectoris is far more frequent (see Chapter XXVII).

(*b*) *Changes in the heart's action.*—This is shown by the peculiar irregularity—the pulsus alternans.

§ 179. **Pulsus alternans.**—A very striking and characteristic sign of exhausted contractility is sometimes seen in the size of the pulse beat. I have repeatedly dwelt upon the fact that the duration and force of contraction depends in a measure on the length of the preceding period of rest. In hearts with good contractility, recovery after a contraction is so rapid that little or no difference can be detected in the size of the beat after pauses of varying duration. In cases of exhausted contractility the matter is very different, recovery being slow, so that the size of the beat has a distinct relation to the length of the preceding diastolic pause. The application of this law assists in the interpretation of many obscure conditions, and gives in many cases a clue to what is going on in the heart. If we observe, for instance, what happens after the long pause following on an extra-systole, we find a different reaction in different hearts. In those with good contractility, the succeeding beat may not be much increased in size, or it may be big. The circumstances causing the increased size are complicated, such as the greater filling of the heart during the long diastole, the lower arterial pressure which the contraction has to overcome, and of course the longer period of rest for the restoration of the contractility. One cannot, therefore, draw any safe deduction from the character of the first beat after the long pause. But the second and third beats may vary remarkably. If the contractility be good the size of the beats after the first big beat are uniform. Sometimes, however, the second beat is smaller than the third and following beats (Fig. 125 A). This difference in the size of the beats is a very important one, as it indicates a very grave exhaustion of contractility. Fig. 125 is a tracing of the carotid pulse of a dog whose heart was dying from exhaustion. An artificial stimulus applied to the ventricle produced the extra-systole r'. The next beat is large, and is followed by a beat, ×, smaller than the succeeding beats. A very similar result is seen in Figs. 125 A, 125 B, and 126 from patients with advanced cardio-sclerosis. Not infrequently, the difference in the size of the beats persists for a shorter or longer period, in such a manner that a large beat alternates with a smaller—the pulsus alternans (Fig. 126). F. B. Hoffman [204] explained this alternating size of beats in the frog's heart as due to an impairment of contractility, and from among the groups of allorhythmia, where every second beat rhythmically varied, Wenckebach [228] distinguished one where a small beat regularly followed a large beat, while the rate of the heart was perfectly

regular. He, most appropriately, desires to limit the name pulsus alternans to this form of allorhythmia. Hitherto the name has been applied very loosely to arrhythmias that we now recognize as due to extra-systoles. The explanation of the cause of pulsus alternans is as follows :—

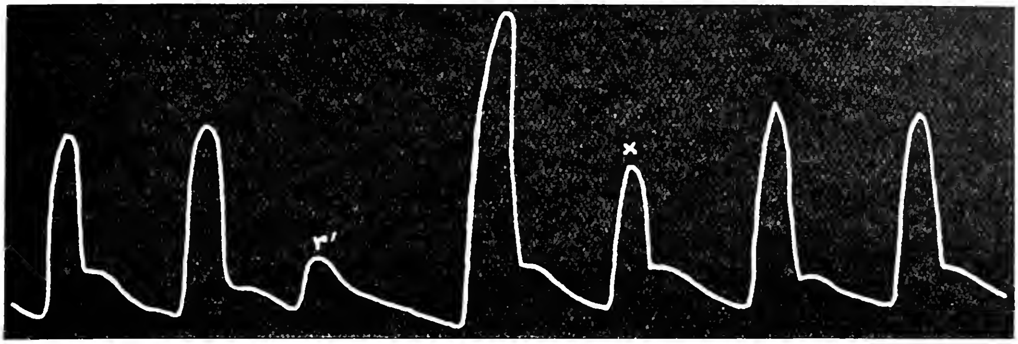

FIG. 125. Tracing from the carotid artery of a dog. The heart was exposed and the ventricle directly stimulated to produce the extra-systole *r′*. The long pause after the extra-systole is followed by a large beat and succeeded by one beat (×) smaller than the other beats. Its small size is due to the shorter period of rest preceding it, and implies grave exhaustion of the contractile power of the left ventricle. Compare with Fig. 125 A (Cushny).

When contractility is depressed, if time be allowed for a full and strong contraction, the longer duration of contraction encroaches upon the period of rest, so that by the time the next stimulus arrives the contractility has not sufficiently recovered, and a smaller and shorter contraction results.

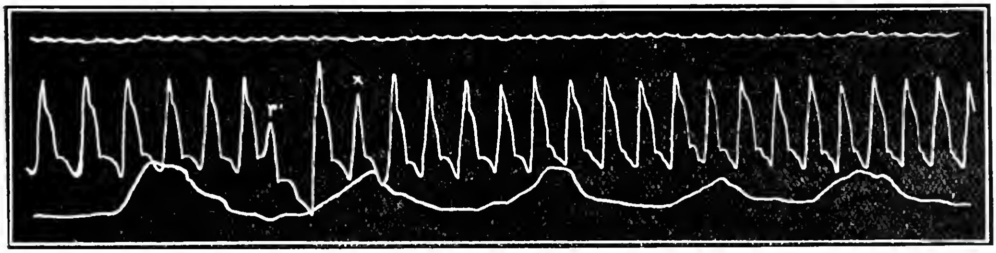

FIG. 125 A. The long pause after the extra-systole (*r′*) is followed by a large beat ; this in turn is followed by a small beat (×), and the succeeding beats are larger. The small beat (×) is an evidence that the contractility of the heart was greatly exhausted. Compare with Fig. 125. (From a case of advanced cardio-sclerosis.)

As this contraction is shorter in duration, the period of rest is thereby lengthened before the next stimulus arrives, so that the contraction will be stronger and longer ; being longer it will again encroach upon the period of rest, and so the process of alternation goes on.

This variation in the duration of the systole can be seen in Fig. 125 A by noting the distance between the up-stroke and the dicrotic notch of the radial beats.

I have now collected a large number of cases in which this irregularity occurs, and it is of far greater frequency than is usually suspected. The

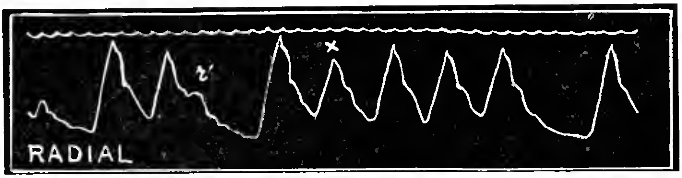

Fig. 125 B. Shows the same variation as Figs. 125 and 125 A. (From a case of advanced fatty and sclerotic heart.)

condition in which it is most frequent is cardio-sclerosis, where it is usually associated with high blood-pressure, extra-systoles, and great limitation of the field of cardiac response, and not infrequently with angina pectoris (Cases 1 and 7, see Appendix I). After a period of rest and a fall of arterial pressure, it may entirely disappear, and it is for this reason that it is so infrequent in hospitals, where the rest in bed favours the restoration of the contractility. In such cases I have frequently been able to bring it back by making the patient hurry up and down a flight of stairs. When it is associated with extra-systoles, an extreme form of irregularity may be presented which seems at first to be hopelessly confused, but by careful analysis we can detect the extra-systole, the long pause after it, and the

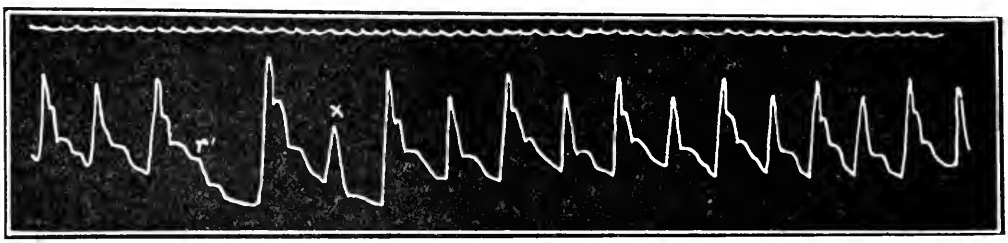

Fig. 126. The alternating character of the pulse is increased after the long pause following the extra-systole (r'); the second beat ($\times$) following the pause appears at the normal interval, but is greatly reduced in size. (From a case of advanced cardio-sclerosis.)

marked difference in the size of the following beats (see Appendix V). In patients who have exerted themselves, a transient but extreme irregularity may sometimes be detected in the pulse. Small beats and big beats may follow one another in a bewildering fashion. But so brief is the duration of the tumultuous action of the pulse, that before one can apply a sphygmo-

graph the arrhythmia has to a great extent disappeared. I have, however, been able on several occasions to catch the irregularity before it had subsided, as in Figs. 127 and 128, in which it may be seen how variable

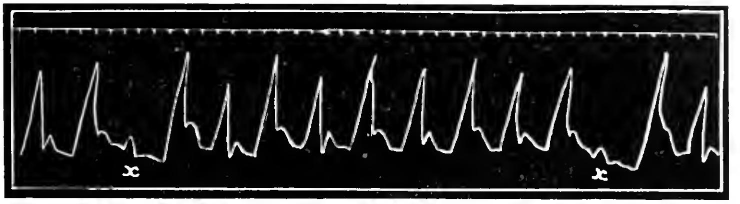

FIG. 127. This tracing and the next were taken from a man with cardio-sclerosis, after walking up a steep road. The pulse felt extremely irregular, and an analysis of the tracing shows it to have been regular in rhythm, but the individual beats varied greatly in force. At x the exhaustion is so great that only a small wave appears in the radial.

is the size of the beats ; one can probably infer that at x x in Fig. 128 the beats were too weak to cause a movement in the radial, so that the pulse is intermittent in character.

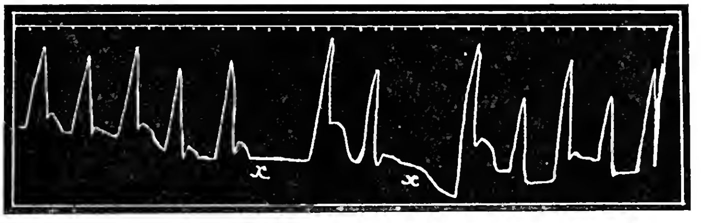

FIG. 128. Intermittent pulse due probably to extreme exhaustion of contractility, the contraction of the ventricle being too weak to send a wave into the radial artery at x. Note the increase of the alternating character of the pulse after the long pause.

It may appear in cases of cardio-sclerosis after an exhausting illness, as bronchitis, when the patient is confined to bed (Fig. 129).

I have found this pulsus alternans during an attack of paroxysmal

FIG. 129. Pulsus alternans, male, 64, cardio-sclerosis and bronchitis.

tachycardia (Figs. 130, and 228, Appendix III), in a case of dilatation of the heart from some obscure cause, in acute febrile affections of the heart (pneumonia and rheumatic fever), and after the administration of digitalis (Figs. 164 and 166).

This form of irregularity must be distinguished from that where an extra-systole alternates with a normal beat (pulsus bigeminus). As has already been described, the latter form of irregularity is caused by the too

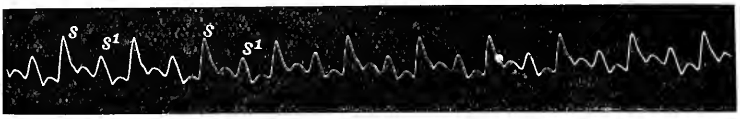

FIG. 130. Pulsus alternans during an attack of paroxysmal tachycardia, sixty-six hours from its commencement. (Case 11, Appendix II.)

early appearance of the smaller beat, which is followed by a long pause. On the other hand, the rhythm in the pulsus alternans is quite regular (compare Figs. 131 and 132), or the smaller beat occurs after a slightly lengthened pause. Volhard [226] has pointed out that this slight delay is due to the fact that the weak contraction takes a longer time to open the aortic valves.

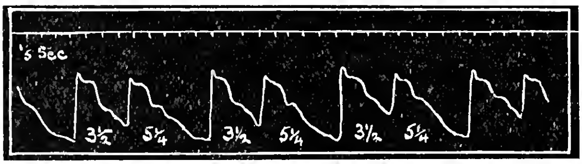

FIG. 131. Pulsus bigeminus, due to the regular occurrence of extra-systoles. The numbers represent in tenths of seconds the duration of each pulse period, and it is seen that the longest pauses occur after the small beats. (Compare with Fig. 132.)

The pulsus alternans can sometimes be perceived by the finger, but it is apt to be overlooked. The sounds of the heart often can be heard to alternate, those accompanying the weaker being less loud. When there

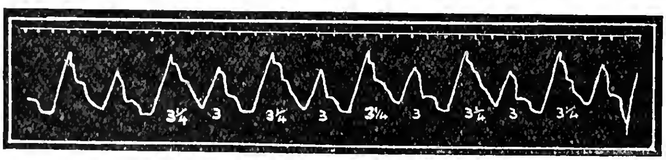

FIG. 132. Pulsus alternans. The numbers show a slight prolongation of the pause before the smaller beat, in contrast to what occurs in Fig. 131.

is a musical murmur the alternation in the strength of the sounds is very marked (Case 24, Appendix V).

§ 180. **Prognosis.**—The prognosis of exhausted contractility naturally depends on the conditions that have induced it. These conditions are

discussed under the different symptoms described elsewhere. It may, however, be pointed out that the pulsus alternans is a very grave sign at all times, and especially in febrile affections. In cardio-sclerosis it implies an advanced degree of exhaustion, and one may confidently infer that the muscular degeneration is fairly extensive, as I have found in a number of post-mortem examinations. A patient may go quietly about for years after the appearance of the pulsus alternans, but it is usually a sign of an irrecoverable underlying condition.

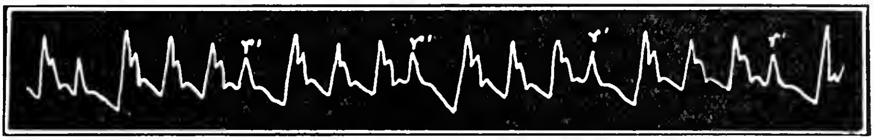

FIG. 133. The extra-systole (r') occurs regularly after every third normal beat. The two beats preceding the extra-systole are of equal size (22. 3. '94).

The grave significance of this seemingly trivial sign is seen in the case from whom Figs. 133 and 134 were taken. The patient was a female, aged fifty, whom I had under treatment for a large thoracic aneurysm about ten years. For some months before her death she showed extra-systoles (Figs. 83 and 84); on March 22, 1894, she had a feverish attack, the temperature 102°, and the pulse-rate 120, still showing the extra-systole after every third normal beat. On the 24th, her pulse-rate and irregularity was the same (Fig. 134), but now it is seen that the second beat (×) after the long pause

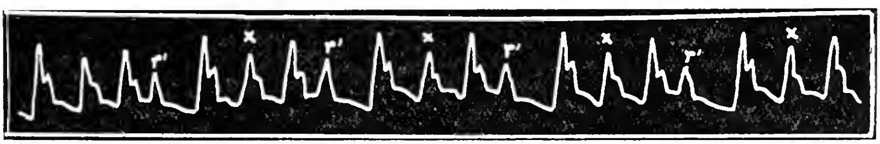

FIG. 134. Shows the same irregularity as Fig. 133, but here the second of the normal beats is always smaller than the third, implying a grave exhaustion of the contractile power of the left ventricle. (Taken 24. 3. '94. The patient died on 26. 3. '94.)

is smaller than the third. She died on March 26, 1894 (see also Case 7, Appendix I, and Cases 23 and 24, Appendix V).

§ 181. **Treatment.**—While the treatment will depend on the conditions causing the exhaustion, we can obtain from the pulsus alternans an idea of the principle which should guide us in every case. Its production by excessive work in degenerated hearts, or after a long period of tachycardia, implies that over-exertion or want of rest is the immediate cause. The occurrence of the larger beat in the alternating pulse shows what rest can do to restore the contractility. Therefore relief from over-exertion, whether

from exercise, rapid action, or from too high blood-pressure, should be the aim. This may be secured by bodily rest, or by the administration of remedies that ensure rest—above all, sleep. When there is a high blood-pressure, dyspnoea, and no dilatation of the heart, chloral has been of great use in my hands. On the other hand, drugs of the digitalis group are not only useless, but may be injurious.

In a few cases I have seen distinct relief from the associated symptoms of pain and breathlessness obtained by large doses of oxygen (p. 278).

CHAPTER XXIII

Dilatation of the Heart (Failure of Tonicity)

Although the matter of the 'tone' of the heart is frequently present in the minds of physicians, nevertheless I venture to doubt if ever it is more than a vague conception. Some writers have described certain conditions associated with it, yet it has not received that consideration its importance merits. The recognition of depression of tonicity will be found to be of the greatest service in appreciating the nature of the heart failure and the remedies appropriate for the restoration of the heart's power. For some years now, I have been inquiring into this function, and although many important features have been revealed, I am far from comprehending its full significance.

§ 182. **The cause of dilatation of the heart.**—Before considering the symptoms produced by depression of tonicity, it is necessary to appreciate the cause of the most prominent of these symptoms, namely, dilatation of the heart, and one cannot fail to be struck with the inadequacy of the explanation usually given for this condition. The prevalent idea seems to be that it is due to an increasing pressure within the chambers forcing the walls outwards. But if it be asked, Whence comes this distending force ? the inadequacy of such an explanation is at once apparent. During systole the increased pressure within the chamber is produced by the contraction of the wall of the chamber itself, and one can scarcely assume that in the process of contraction dilatation is produced. Dilatation of an auricle,

it is true, might be produced by the forcible regurgitation of blood from a powerful ventricle, but such a thing can only happen when there is a lesion of the auriculo-ventricular valves. Regurgitation, apart from valvular lesion, can only occur after the muscle-fibres surrounding the auriculo-ventricular orifices have become relaxed—that is to say, dilatation of the auricle from such a cause would be produced after dilatation of the ventricle.

That neither the resistance opposed to a chamber during the systole, nor the distending force during its diastole, is the cause of dilatation becomes evident when the conditions observed in certain hearts are carefully studied. Thus hearts whose walls are thinned, and whose muscle-fibres are degenerated, may continue to work against an abnormally high arterial pressure, and never show any signs of dilatation. In fact, the wall of the left ventricle may be so thinned that it actually bursts in its effort to overcome the aortic pressure, yet the walls show no signs of dilatation. Professor Keith, who has especially looked into this matter of ruptured hearts, informs me that such hearts may show no sign of increase in the size of their cavities. In a patient under my care the heart was so enfeebled that he could scarcely walk fifty yards without an attack of angina pectoris coming on, but no enlargement of the heart could be detected. He died suddenly from rupture of the heart, and I found that part of the heart-wall was so thinned as to be made up of little except the endocardium and pericardium, yet, notwithstanding this enfeeblement of the heart-wall, there was no sign of dilatation of the cavity.

Dilatation of the left ventricle may occur even when the diastolic force filling the ventricle is greatly diminished, as in cases of pure mitral stenosis. Here the quantity of blood reaching the ventricle and the force with which it enters the ventricle are so greatly diminished that we must look for some other cause for the dilatation of the left ventricle that is found in advanced cases of mitral stenosis.

§ 183. **The function of tonicity.**—In default of this mechanical explanation we turn naturally to the functions of the normal heart, to inquire what maintains the fibres in health in a position short of their extreme relaxation. In this way we may succeed in obtaining a more definite conception of what occurs in dilatation, even if we fail to elucidate altogether its etiology.

This function of maintaining a position short of extreme relaxation is not peculiar to the cardiac muscle, but is also met with in the ordinary skeletal fibres, and in both cases it is due to the possession by the fibres of the function of tonicity.

§ 184. **The symptoms of depression of tonicity.**—These symptoms

are threefold: (1) those due directly to the changes in the heart, viz. increased size of the heart, alterations in the position and in the character of the movements of the heart, and the presence of murmurs; (2) those associated symptoms due to the failure of the circulation in remote organs and tissues, as dropsy, enlargement of the liver, and breathlessness; (3) certain reflex sensory symptoms mainly shown by regions of hyperalgesia affecting the skin, breast, and muscles of the left chest and axillary fold, and sometimes also the left sterno-mastoid and trapezius muscles.

§ 185. **Dilatation of the heart.**—The evidence of dilatation of the heart is made out by marking out the increased size of the heart. I need not dwell upon how this is done, for the methods for percussing out the heart's dullness are described in sufficient fullness in every handbook of physical diagnosis. For practical purposes the transverse dullness at the level of the fourth interspace gives on the whole the best estimate of the size of the heart. In exceptional cases the whole area of deep dullness may be with advantage mapped out, as when dullness is found extending to the left above the third rib. In such cases the possibility of pericardial effusion should be kept in mind.

It is very difficult to tell with certainty what share each chamber of the heart takes in the production of the increased size, on account of the displacement of the whole organ. The manner in which the heart is fixed above, by the aorta, pulmonary artery and veins, and superior vena cava, and below by the inferior vena cava, keeps fixed an axis on which the heart to a certain extent rotates in the enlargement of its various cavities. The tendency when the right ventricle dilates, is for it to push the left ventricle to the left and behind, with the result that in the great majority of cases we get evidence of an extension of the dullness to the left. When the right auricle becomes greatly distended, it may push itself to the front of the chest, and, as Keith's dissections show, compress the right ventricle to a remarkable degree. When there is extension of the dullness beyond the right border of the sternum, it may with certainty be put down to the right auricle, except in aneurysm or other intra-thoracic tumour.

The manner in which the right heart pushes over to the left side is well brought out in Figs. 135 and 136. These are typical of the dilatation secondary to mitral stenosis. If a comparison be made with the position of the chambers in the normal heart (Fig. 19), it will be observed that the increase to the right side, notwithstanding the great dilatation of the right heart, is very slight, whereas the great increase in the size of the heart is to the left—sometimes with only a slight depression of the apex (Fig. 135), sometimes with considerable depression (Fig. 136). Note also how the

right auricle pushes into the second left interspace. So long as the lungs cover a portion of the heart the perceptible movements will be entirely due to the right ventricle; but when the lung is pushed from the front of the heart, then the real apex will be found at the extreme left (see § 83).

In seeking for the cause of the increased dullness, the character of the impulse should always be studied, not only to determine the nature of the

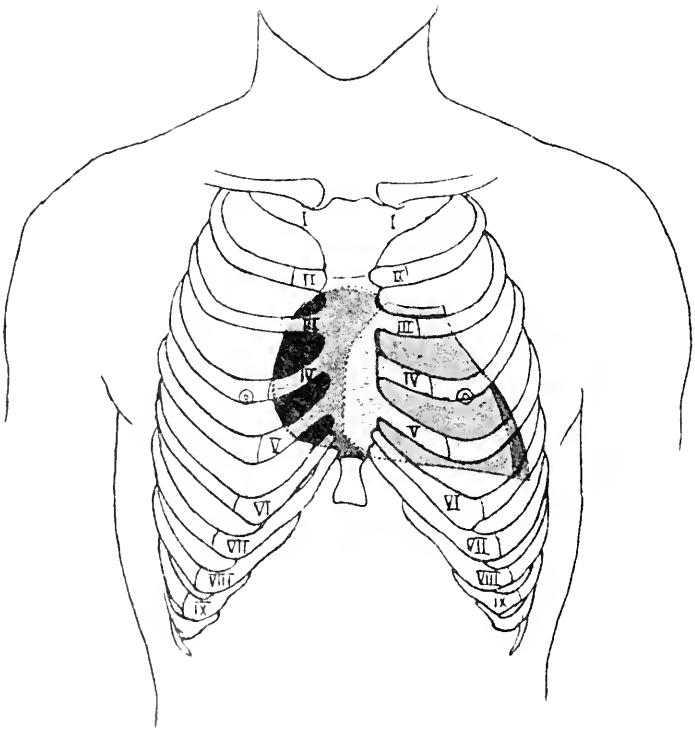

FIG. 135. Position of the chambers of the heart in extreme dilatation, as in the late stage of mitral stenosis. The portion of the heart on the right of the sternum shaded deeply together with that behind the sternum up to the dotted line represents the right auricle, while the small strip to the left shaded deeply represents the left ventricle. The part between is the right ventricle. (Harris).

heart's enlargement, but to distinguish it from pericardial effusions and displacement of the heart by such conditions as aneurysm, pleural effusion, and so forth. Another point to bear in mind is that, in the early stages of enlargement of the heart, the lung may still cover a part of its left border, but with persistence of the enlargement the lung is compressed, and if non-adherent, recedes from the anterior surface of the heart, altering altogether the character of the apex movements.

§ 186. **The cause of functional murmurs.**—Functional murmurs

have hitherto been looked upon as a consequence of simple dilatation of the heart, but this explanation is far from being sufficient. Thus we may have considerable dilatation of the heart without a murmur. Again, we may have very little dilatation with marked systolic murmurs at apex and base, and with a great regurgitant wave in the veins. The explanation of these apparent anomalies seems to be in the condition of the muscles supporting

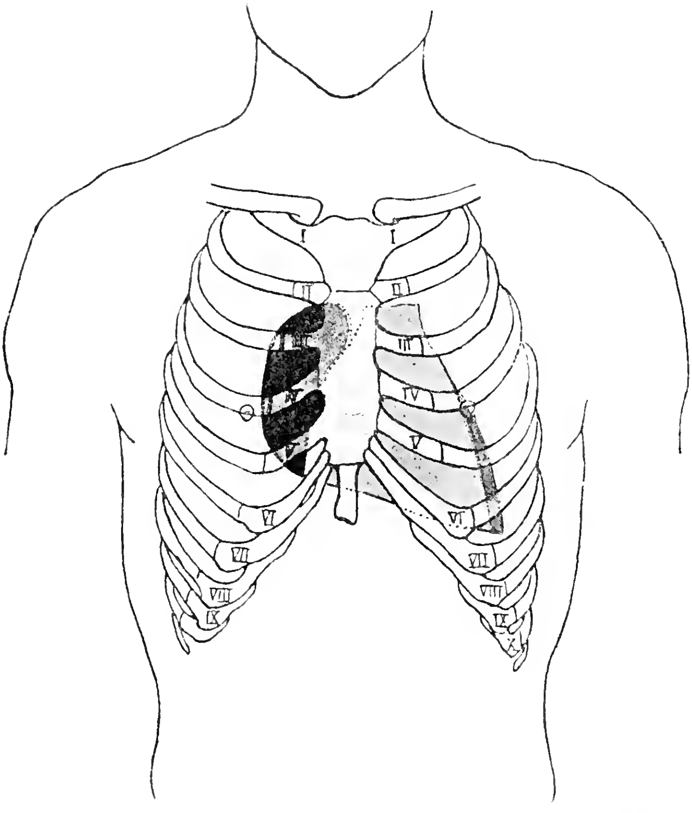

FIG. 136. Dilatation of the heart with the apex beat displaced downwards. The shading is the same as in the preceding figure.

the auriculo-ventricular orifice. If their tonicity be depressed regurgitation ensues, and gives rise to the functional murmurs.

§ 187. **The consequences of dilatation of the heart, and how they are brought about.**—Before describing in detail the results that follow dilatation of the heart, it is necessary to consider the manner in which these symptoms of heart failure are brought about. The maintenance of the circulation is due to the contractile force of the heart, and normally the factors concerned are so balanced that everything is done to facilitate

the work of the heart. It is reasonable to assume that the chambers of the heart are normally of the size that enables them to contract with the greatest efficiency. The dilatation of these chambers will therefore embarrass the heart muscle, with the usual result, a limitation of the reserve force. At first, this limitation may only call forth disagreeable symptoms when it is exhausted, the exhaustion occurring much sooner than normal. The degree of exhaustion depends on the integrity of the heart muscle. If the contractile force is embarrassed by inherent defects of the muscular wall, or by other causes, such as irregularity of action, or valvular defects, then the heart failure corresponds to the degree of embarrassment and inability of the muscle to overcome it. For this reason we find all degrees of heart failure associated with dilatation. In the milder cases there may be only the subjective symptoms of breathlessness, palpitation, and weakness. In the more extreme cases, dropsy (more or less extensive), diminished secretion of urine, effusion of fluid into the serous cavities, enlargement of the liver, lividity of the face may be present. The rationale of the production of this extreme heart failure seems to be as follows : So long as the contractile force of the heart is able to maintain a degree of arterial pressure sufficient to supply the organs and tissues, the heart failure will be limited to those subjective symptoms involved in a great reduction of reserve force ; the patient is comfortable at rest, for the heart is then able to maintain the circulation, but is distressed by exertion, for the heart has so little reserve force that it is unable to meet the extra demand. When the force of the heart fails to maintain the arterial pressure at the height necessary for the tissues, then we get the symptoms in the remote organs and tissues (dropsy, ascites, enlarged liver, &c.).

I have endeavoured in a number of ways to demonstrate the relationship between dilatation of the heart and these evidences of extreme heart failure, and I give a few instances where the association of these symptoms with dilatation of the heart is clearly marked.

In advanced cases of cardio-sclerosis with a blood-pressure continuously high, between 180 mm. Hg. and 200 mm. Hg., the heart may be of normal size, or only very slightly enlarged. There is great limitation of the field of response, exertion readily inducing attacks of angina pectoris or breathlessness. Cheyne-Stokes respiration may occur also, and attacks of cardiac asthma or severe dyspnoea at nights. There may be large pulsation in the veins of the neck, but no dropsy. Patients may gradually weaken and die, and the heart remain unchanged in size. On the other hand, in the course of a day or two, one may be struck by a great change. The patient seems easier ; the blood-pressure has fallen to 150 mm., or lower ; attacks of angina

pectoris, cardiac asthma, and Cheyne-Stokes respiration disappear. But the legs begin to swell, the urine becomes scanty, the jugular pulse disappears, the breathing is continuously hurried, and the patient has to be propped up in bed. He may begin to expectorate blood-stained mucus, and there is evidence of oedema of the base of the lung. If the heart be examined, it will be found to extend one or two inches further to the left, and it may be a mitral murmur has developed.

Even more striking, because more sudden and violent, are the changes that take place in certain cases of paroxysmal tachycardia. In describing this condition (Chapter XX), I pointed out that the symptoms varied in patients according to the condition of tonicity,—if the heart remained unaltered in size during an attack, the symptoms were less marked, and the condition less grave than if the heart dilated. In the cases recorded in the Appendix (Cases 11 and 12, Appendix II), the heart dilated, and in the course of a few hours symptoms of extreme heart failure set in. I have seen these cases on several occasions shortly before an attack, and watched the steady progress of the change. The hearts were nearly normal in size, but in three hours' time the transverse diameter had increased by two inches, the face had become livid and the lips swollen. The veins of the neck, which had shown little movement, now pulsated largely in the nodal rhythm. In the course of twenty-four hours oedema of the legs appeared, and the liver became large, and in one case pulsated. After some days the dropsy extended up the legs, the abdomen became distended, and the urine scanty. With the cessation of the attack of paroxysmal tachycardia, the patients at once experienced relief, and in a few hours every vestige of heart failure had disappeared, and the heart itself returned to a normal size and rhythm. I give these instances because, owing to the sudden change of appearance, one could account fairly satisfactorily for the symptoms. In various modifications they will be found associated with extreme heart failure from all forms of cardiac disease. I have seen many cases in which the inception of the nodal rhythm was followed by these changes, and when it persisted a partial recovery only followed, with reduction of the size of the heart. In certain alcoholic hearts—especially in those of Graham Steell's group of 'muscle failure'—these phenomena can also be recognized, as well as in other conditions.

This manner of looking at dilatation, and the cause of dropsy and other symptoms, has a very practical bearing on the treatment of heart failure associated with valvular disease, the nodal rhythm, and cardio-sclerosis.

§ 188. **Dropsy.**—Oedema of the subcutaneous tissues is a common feature in heart failure with dilatation. It is apart from my purpose to

discuss the various theories propounded to account for its occurrence, it being sufficient here to note that its appearance is often a definite sign of heart dilatation, and its disappearance an equally definite sign of restoration of the heart's tone. It begins first in the most dependent parts : in people not confined to bed it is found first about and above the ankles ; in people lying in bed, across the sacrum. It may linger in the legs for years in some folks—worse towards night, better in the morning. In extreme cases it invades the thighs and abdominal wall. The loose cellular tissue of the scrotum and the penis and vulva becomes infiltrated, and may attain an enormous size. Before marked effusion takes place into the abdominal cavity, the bowels often become greatly distended. It may finally invade the pleural cavities, producing hydrothorax. The distended abdomen and the hydrothorax add to the embarrassment of the breathing. If the patient leans more to one side than to the other, in extreme cases, the arm and check of that side may become greatly swollen. When an arm becomes swollen apart from this, and where there is not extensive oedema, one may suspect a clot in some of the larger veins in or near the chest.

Associated with dropsy there is usually a diminished urinary secretion, and disappearance of the dropsy usually coincides with an increased flow of urine.

The significance of oedema is extremely varied. Many elderly people, especially if they are stout, may for years have their legs more or less swollen even if their hearts present no particular abnormality beyond a slight dilatation, though it is more common amongst those with the nodal rhythm. It may be present in attacks of heart failure to an extreme degree, with ascites and hydrothorax, and notwithstanding the patient may make a good and lasting recovery. These are found more particularly in cases of rheumatic affection of the heart of some duration, starting in some with the nodal rhythm. If the heart reverts to its normal rhythm, the disappearance of the dropsy is more speedy than its onset. If the heart reacts to digitalis, the disappearance of the dropsy accompanies the other beneficial effects of the drug. When all attempts to restore the heart fail, the dropsy increases, embarrasses the heart and the respiration by effusion into the serous cavities, and adds much to the suffering of the patient, who drifts to a fatal issue.

§ 189. **Enlargement of the liver.**—Another result of the failure of the circulation secondary to dilatation of the heart is swelling of the liver from passive congestion (Chapter XIV). It may not appear in the earlier stages in the first instance, but when a patient has once recovered from an attack of heart failure with enlargement of the liver, every subsequent attack induces this symptom, sometimes before any sign of dropsy sets in.

There may be associated with the enlargement a certain amount of jaundice, and the combination of enlarged liver and jaundice, with the wasting that sometimes accompanies long-continued heart failure, may raise the suspicion of malignant disease of the liver (§ 121). The dilatation or irregular action of the heart should direct attention to the real nature of the trouble.

There may be a considerable degree of pain and discomfort associated with the enlargement of the liver, and the painful contracted muscles may embarrass the respiration (described in § 117).

§ 190. **Oedema of the lungs.**—A symptom of great value is found in the careful auscultation of the bases of the lungs in cases threatened with some forms of heart failure. I carried out for some years an extensive observation on all kinds of people—healthy, and with failing hearts from a great variety of causes—and I was able to anticipate attacks of heart failure in a great number of cases. If one systematically examines the bases of the lungs in elderly people who are perforce confined to bed, as in consequence of an operation or a fractured leg, in a certain proportion the earliest symptom of heart failure will be found in the appearance of fine crepitations at the bases of the lung. The same experience will be met with in patients confined to bed from any exhausting complaint, particularly if the heart muscle be involved in the ailment, as in typhoid fever. Many patients with mitral lesions have no dropsy, but suffer from severe attacks of heart failure with great breathlessness. In such cases the bases of the lungs will be found to show signs of oedema at the early stages of the breakdown.

It has been my habit in these cases to begin the examination of the patient by asking him on which side he lies, then make him sit up, and while I auscultate the base of that lung on the side he had lain, I ask him to take in one full and deep inspiration. This opens up the alveoli at the base, and if there is any abnormal moisture it is manifested by numerous fine crepitations. Healthy people show no sign of this. Slightly weakened hearts may show it with the first deep inspiration only ; if there is distinct cardiac enfeeblement the crepitations do not disappear at first, but persist. I have seen cases where the first sign was the crepitation during the first deep inspiration, and gradually the crepitations became more persistent until the resonance of the bases of the lung became impaired, even to complete dullness, with no breath sounds, and at the post-mortem examination the lungs at the bases have been sodden and airless. In some instances there have been patches of inflammation (catarrhal pneumonia—the hypostatic pneumonia of the feeble).

I have also seen this hypostatic congestion disappear, and as the patient improved the crepitations gradually disappeared, the last sign being the crepitations with the first deep inspiration.

I have found this method of observation of the greatest practical use. In the elderly it governs the position which the patient should occupy—lying down or propped up. In typhoid fever it is a prognostic sign of the very greatest value—the absence of oedema indicating that the heart has escaped infection ; its presence and gradual increase, a sign of great gravity. In heart disease it is likewise one indication of the heart's condition, and in the complication of pregnancy and heart disease it is one of the most important guides in the management of these cases.

It is of no less importance in treatment, as will be realized when the reason for its appearance is appreciated. It invariably accompanies dilatation of the right heart, and the manner of its production is as follows : The factors that move the blood through the lungs are twofold—first and most important, the right ventricle ; and second, the movements of respiration. In healthy hearts the first of these is so powerful that the second is scarcely appreciated. When, however, the right ventricle is enfeebled, the assistance of the respiratory movements becomes necessary. When the patient lies in bed on one side, the pressure of the ribs on the mattress restrains their movement, so that the flow of blood through this part of the lung is retarded, and oedema results. This can be shown in the early stages, for when the patient breathes deeply the whole of the crepitations may disappear.

From this account will be realized the part that can be played in suitable cases by placing the patient in a position to breathe freely, avoiding the restraint exerted by pressure on the ribs, and by making the patient deeply inspire. In addition, the importance of recognizing the nature of the symptoms due to the enlargement of the liver is here apparent, where not only the abdominal muscles, but the intercostals also, may be tender and contracted. In thus ceasing to act as respiratory agents, while exercising their primitive function of protection, these contracted muscles further add to the embarrassment of the heart in its work.

§ 191. **Urinary symptoms.**—I doubt if ever we get the characteristic urinary symptoms of heart failure in the absence of dilatation of the heart. These symptoms are a scanty secretion and increased specific gravity, and frequently the presence of albumen. A diminished supply of blood to the kidneys may cause a large quantity of albumen to appear in the scanty urine, as can be observed in heart-block and nodal bradycardia, when the heart's rate becomes very infrequent. The diminution of the quantity usually goes hand in hand with the dropsy. The cause in the main is

a fall in arterial pressure and a rise in the venous, with consequent venous stasis in the kidneys. Other conditions may co-operate, such as the chemical constitution of the fluid in the tissues and changes in the secreting cells of the kidney. It is often a difficult point to determine whether the albuminuria has been pre-existent, or whether it is induced by the venous stasis and subsequent inflammatory changes in the kidneys. The history of the patient will help, and the presence of arterio-sclerosis and retinitis point to a pre-existent Bright's disease. It may be necessary to suspend judgement until a recovery of tonicity of the heart restores the circulation, as with the increase in flow of urine the albuminuria may entirely disappear.

It is often useful to direct the patient's attention to the urinary secretion, as its diminution may give the first warning of an impending breakdown, and the increase in the flow is often the first sign of recovery of heart power.

§ 192. **Prognosis.**—While the degree of tone present is of first-class importance in estimating the nature of the heart failure, we must use great discretion in determining the value of dilatation as a prognostic factor. Great dilatation may be compatible with a favourable prognosis, while a much smaller degree of dilatation may be a sign of considerable gravity. If we find the patient getting along comfortably and well with a dilated heart, it means that the muscular tissue is otherwise sound, and that there is no material obstacle to the work of the heart, and the muscle itself is able to overcome the embarrassment induced by the dilatation.

Another element in the prognosis is the manner in which the heart responds to treatment, especially to drugs of the digitalis group. As I shall point out in Chapter XXXIV, tonicity is one of the functions of the heart which digitalis readily affects in many cases, and it is probably because of the effects of this drug on this function that it has often such a wonderfully beneficial influence on the heart. The improvement produced by digitalis is therefore a guide to the condition of the heart muscle, and to the susceptibility of this function to its action. On the other hand, when dilatation of the heart and the symptoms accompanying it described above are unresponsive to digitalis, the outlook becomes grave. Especially is this the case in the heart failure with dilatation in advanced arterio-sclerosis.

§ 193. **Treatment.**—As dilatation is invariably secondary to some other condition of the heart, the treatment has to take into consideration other factors. I may point out, however, that, unless in the acute febrile stage, dilatation is an indication for the prescription of digitalis in all rheumatic hearts. In other conditions it should be tried, especially if there is dropsy and deficient secretion of urine. The treatment of such

associated symptoms as dropsy and diminution of urine should consist in the attempt to restore the heart's strength. When dropsy becomes a distressing symptom, special means have to be undertaken for its removal. As free diuresis is the most effective way of getting rid of it, a great many agents are recommended which effect this purpose. The virtues of many a prescription can be established by the recital of illustrative cases in which its exhibition was followed by an extraordinary discharge of urine and speedy disappearance of all dropsy. These preparations will be found to vary from the mixtures containing every conceivable drug that is supposed to have diuretic properties, to some recent synthetical preparation. The fact of the matter is, that in many of these cases the secretory activity of the kidneys seems to be in temporary abeyance, and some slight adventitious aid gives it the necessary stimulus. This auspicious moment coinciding with the administration of the drug, results in a profuse diuresis. This aid need not be a drug. I have seen a patient, who had to sit up in a chair for three weeks on account of his breathing, become extremely dropsical, passing very little urine. The mere return to bed was followed at once by a profuse diuresis and the rapid disappearance of the dropsy. It is, however, necessary in many cases to try various agents, and happily here the digitalis group is most effective, the combination of digitalis, squill, and calomel being particularly useful, not only from its action on the heart and kidneys, but also from its effects on the bowels. When these drugs fail others may be found to act, such as theobrominae sodii salicylas (Diuretin) or theocin-sodium acetate. In some cases the elimination of common salt from the food helps to reduce the dropsy. In all cases of dropsy the bowels should invariably be kept well moved.

Special efforts to give relief are often necessary. In certain cases when the patient can go about, an elastic bandage skilfully applied is beneficial, particularly in those hard, swollen legs when the skin threatens to give way. Massage also is of assistance. When the legs or genitals become greatly distended, deep pricks with a needle will often be followed by a free flow of serum and a great diminution of the swelling. The utmost cleanliness should be observed in carrying out this simple procedure. The employment of Southey's tubes inserted into the legs or abdominal cavity will often drain off a large quantity of fluid. The abdominal and thoracic cavities may need to be tapped ; such tapping invariably gives temporary relief. In some advanced cases where the penis is greatly swollen (ram's horn), there may be inability to void urine. In using a catheter there may be some trouble in finding the meatus, the glans penis being buried in the scrotum under the swollen prepuce. If the swollen foreskin be gently but

firmly grasped in the hand and compressed, the fluid is driven out, and the glans can then be exposed.

The employment of judicious breathing exercises in oedema of the lung is often beneficial, the patient sitting up and breathing slowly and deeply. In severe cases this is limited at first to a few movements. If the patient bears the exercise, these deep respirations should be employed at regular intervals every two or three hours when awake. In the intervals the patient should be propped as high as he can bear with comfort.

CHAPTER XXIV

Acute Febrile Affections of the Heart

§ 194. **Manner in which the heart is affected in fever.**—In considering the state of the circulation in febrile conditions, it is necessary to bear in mind three facts, viz. that the heart's action is modified by an increase in temperature, that the heart reacts differently according to the toxins produced by the agent causing the fever, and finally, that the heart itself may be the seat of the conditions causing the fever. Recent researches demonstrate conclusively the invasion of the heart by the specific organism in rheumatic fever, pneumonia, typhoid fever, diphtheria, erysipelas, influenza, and various septic infections. The results of such invasion are shown in the occurrence of endocarditis, myocarditis, and pericarditis. The symptoms evoked in such invasions are not always distinctive, and may resemble the symptoms induced in the heart by febrile conditions alone, or by toxins produced from other sources in the body. I dwell upon this because an attempt should always be made in febrile conditions to judge rightly the effects on the heart. One cannot but be struck, for instance, by the fact that people with previously seriously damaged hearts may pass scatheless through severe attacks of pneumonia or typhoid fever, while the young and vigorous may succumb after a few days' illness on account of the implication of the heart in the disease.

Another point to bear in mind is that in the invasion of the heart the specific organism rarely affects one tissue alone. In order to be exact and

methodical, writers usually describe separately the symptoms of endocarditis, myocarditis, and pericarditis. But if one reflects on the nature of the symptoms, such as the condition of the pulse, its strength, rate, and rhythm, the size of the heart, and the praecordial distress—symptoms which are usually included in the description of endocarditis and pericarditis—it will be realized that they are not really the manifestations of endocarditis or pericarditis, but are the signs of a myocardial affection. One must consider carefully the murmurs arising in the course of a febrile attack, even in rheumatic fever, for the presence of a murmur may not necessarily mean the invasion of the mitral valves by the inflammatory process, but may be due to the tonicity of the poisoned heart muscle failing and giving rise to incompetence of the mitral orifice—due, therefore, not to an endocardial, but to a myocardial affection. Endocarditis and pericarditis, both acute and chronic, bulk so largely in medical literature only because an abnormal sound invariably impresses the mind more than an abnormal sign perceptible by the other senses, and the easy recognition of the valvular murmur and the friction sound has led to the associated symptoms being ascribed to the same lesion.

§ 195. **The febrile heart.**—By this term I mean the changes induced by the rise in temperature. The whole circulatory apparatus is remarkably sensitive to alterations in temperature, whether arising from external sources or due to changes within the body. The most striking of these is the change in rate—a rise in temperature increasing the rate, and a fall diminishing it. In the more simple febrile affections there is a certain correspondence between the height of the temperature and the rate of the heart's contraction. Roughly speaking, there is an increase of from eight to ten beats with a rise of temperature of one degree F. This does not hold universally, but any considerable departure from this rule should always arouse watchfulness and suggest the possibility of other complications, such as the involvement of the heart in the infection.

In the simple febrile heart the radial artery enlarges and the pulse remains of good strength, particularly during the diastolic phase of the cardiac cycle. The heart itself shows little change at first, beyond having its rate abnormally accelerated by exertion. The sounds are clear and distinct, and there is no increase in size. With long continuation of the fever a certain amount of dilatation may arise. This most readily occurs on the right side of the heart, particularly if the pulmonary circulation is interfered with, as by a pneumonia or a pleuritic effusion, or by lying a long time on the back, as during the course of typhoid fever. The sounds may become feeble, or systolic murmurs may develop at the tricuspid and mitral orifices, and the

characteristic pulsation of the right heart in the epigastrium become visible (Figs. 29 and 30, pp. 84, 85). In many of the minor febrile attacks the course is modified by the nature of the toxins generated. A rise of a few degrees of temperature may provoke an undue frequency—120–140, and with the subsidence of the temperature the rate may speedily fall to normal, leaving no ill effects. A slight rise of temperature may even be accompanied by a fall in the pulse-rate—sometimes, if the patient's pulse is normally a slow one, below fifty per minute. I have found remarkable variation in rate with the same temperature at different times, in the same individual, due probably to a difference in the agent producing the fever. The effects produced by agents other than a rise in temperature cannot perhaps be better illustrated than by those occurring during an attack of ague ; here in the course of twenty-four hours, with a continuously high temperature, we have a remarkable series of changes in the pulse. During the cold stage the pulse becomes small and scarcely perceptible, on account of the contraction of the peripheral arteries. The blood driven from the surface and from the arterial system accumulates in the venous system and in the internal organs. Then the lips and fingers become blue, and the congestion of internal organs may reach such a degree that capillary haemorrhages occur within them. Within a few hours, the temperature still being high, the arterioles relax, the arteries become larger, and the pulse itself is of considerable force.

§ 196. **Acute febrile affections of the heart.**—The lesion induced by the invasion of the heart by specific organisms is rarely limited to one structure or tissue, so that it would be better to use the term carditis than to employ such misleading terms as endocarditis and pericarditis. This will be brought out more clearly when an analysis is made of the symptoms present in any given case, and I shall endeavour briefly to summarize the symptoms and try to apportion them to the particular tissues affected. I do this here, because by the appreciation of the nature of the primary lesion we are better able to understand the conditions found many years after, when the cicatrizing process has wrought other changes.

§ 197. **Symptoms in myocarditis.**—The rate and rhythm are the most easily recognized. Seeing that a rise of temperature alone induces an increased frequency, it is impossible to apportion the relative influences of this and of the myocardial infection. But many cases of moderate fever have a heart-rate greatly accelerated, and then we can infer there is some other factor at work than the rise in temperature. It would be of great interest to know the mechanism by which increased rate is brought about by myocardial lesions, whether through nerve stimulation arising reflexly from the inflamed tissue, or from the increased irritability of the

muscle, particularly of the tissue in which the stimulus for contraction arises. But here such an inquiry would be purely speculative.

Changes in rhythm come under a different category, and in irregularity we often get a clue to the changes that are going on in the muscle. The arrhythmia due to purely nervous influences is generally abolished during the excitation of the heart by the fever—the chief exception being the arrhythmia due to vagus stimulation in affections of the brain (as in tubercular meningitis).

The myocardial irregularities have not been as carefully worked out in acute conditions as their importance demands, and the slight advance I have made in the study of the subject shows it to be of the utmost importance if we would understand the pathology of the living heart.

Depression of conductivity.—The most characteristic evidence of direct damage done to the muscle of the heart is the irregularity or pulse intermission due to the dropping out of ventricular systoles, because of the damage done to the a.-v. bundle by the lesion. In carefully watching cases of rheumatic fever, I have detected the appearance of a mitral murmur, and found a lengthening of the interval between the auricular and ventricular systoles, followed later by the presence of an aortic diastolic murmur. The administration of digitalis in such cases has produced a mild form of heart-block (Case 27, Appendix VI). This affection of the a.-v. bundle, shown by slight spontaneous heart-block, I have detected in rheumatic fever and in influenza (Fig. 119), and it has been detected in other acute affections. Professor Holst, of Christiania, has shown me tracings of mild heart-block from a case of septic poisoning, and Dr. Grosh, of Toledo, U.S.A., similiar tracings from a case of puerperal fever, while Dr. Cooper, of San Francisco, has shown me tracings of complete heart-block from a case of septic poisoning under the care of Dr. Jellinek. In this last case the patient died within fourteen days of the onset of the heart-block, and an examination of the heart by Dr. Ophuls showed an anaemic necrosis of the muscular septum involving the a.-v. bundle, consequent on a recent thrombus of the nutrient artery.

In acute affections of the heart, writers usually content themselves by mentioning irregularity as one of several symptoms, but the instances cited show that if graphic records were taken this condition would be found fairly frequent.

Depression of contractility.—Towards the later stages of fatal pneumonias, I have frequently detected a missed beat or an irregularity. Analysis of a number of these cases has shown that it has probably been due to failure of the power of contraction. Thus a typical example is seen

in Fig. 137, where towards the end of the tracing there is shown a marked pulsus alternans. The missed beats in the earlier part of the tracing I think are due to the contraction having been so feeble that it failed to propagate a wave into the arteries. Thus we see in Fig. 138, from another case of

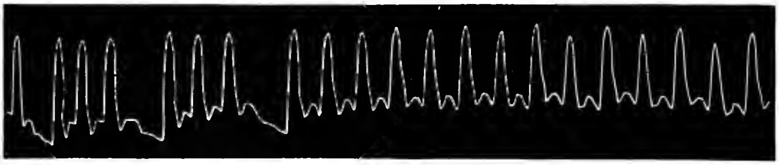

Fig. 137. Irregular pulse in the course of a pneumonia, showing the pulsus alternans in the latter half of the tracing. The intermissions may be due also to failure of contractility.

pneumonia, how the small beats s' arise at the normal interval, but the exhaustion of the heart is so great that it only sends out a small quantity of blood, and after the last small beat it fails altogether to send out a wave. Such signs in pneumonia I have found always to be of the gravest signi-

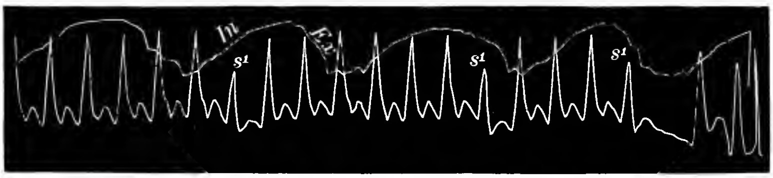

Fig. 138. Tracing of the respiratory curve and of the irregular pulse in the course of a fatal case of pneumonia. The small beats s^1 are probably due to exhausted contractility.

ficance. John Hay has shown a similar arrhythmia in a patient suffering from septic poisoning.

Extra-systoles.—Extra-systoles are of rare occurrence in severe infectious febrile hearts, but Fig. 139 shows the occurrence of extra-systoles

Fig. 139. Extra-systoles occurring in the course of a fatal attack of rheumatic fever.

in the course of a fatal attack of rheumatic fever. As there is no compensatory pause, the premature beat is probably of auricular origin (§ 145).

Nodal rhythm.—I have seen in several instances the sudden inception of this rhythm in pneumonia, and always with disastrous results. In one patient whom I saw in consultation everything seemed to be progressing favourably, but while talking to the doctor in attendance we were suddenly

summoned to the bedside of the patient, and found that the heart's action had, in the interval since our seeing him, taken on this nodal rhythm, and the pulse had become rapid and irregular. The patient died a few hours after. In another case I was called to see, the doctor told me the patient had passed well through an attack of pneumonia terminating by crisis. The day following the fall of temperature the patient suddenly became weak and ill, and the doctor being summoned found him collapsed. I saw him shortly after and found the heart had taken on the nodal rhythm ; the doctor assured me that he had gone through the pneumonia with a good and regular pulse. He also died a few hours after.

Dilatation of the heart.—Evidences of myocardial affections can be found in the changes in the size of the heart. The symptoms of dilatation have been fully described in Chapter XXIII, but it is well to remember that the size of the heart may very speedily become greatly increased in the course of a febrile affection of the heart, as in rheumatic fever, diphtheria, &c. In such cases the sounds often become very faint, and soft murmurs may arise at the mitral and tricuspid orifices, and simulate valvular changes.

§ 198. **Symptoms in endocarditis.**—The only direct evidence of acute endocarditis is the presence of murmurs at one or other orifice of the heart. For practical purposes there are only two murmurs which need to be considered here, viz. a mitral systolic murmur and an aortic diastolic. It is not always easy to tell whether the appearance of a murmur during a febrile attack is due to involvement of the mitral valve in an endocarditic process or to the relaxation of the muscle supporting the orifice. A diastolic aortic murmur is, as a rule, diagnostic of the involvement of the aortic valve in some destructive process. At first this murmur is so faint that one is simply conscious of the fact that the sound does not end with sufficient abruptness. Gradually, however, this passes into a very soft short whiff at the end of the second sound, becoming day by day more marked.

In the vast majority of cases the murmurs due to endocarditis are not definitely recognized until some time after the subsidence of the fever—when the sclerosis sets in. This is particularly the case with presystolic mitral murmurs, which are never recognized during the acute condition that induces the lesion, unless there is narrowing of the mitral orifice on account of vegetations. The formation of vegetations at the mitral and aortic orifices may give rise to murmurs indistinguishable from those due to destruction of the cusps. The presence of a musical murmur may generally be assumed, particularly in acute cases, to be due to a vegetation. An attack of hemiplegia during an acute febrile condition may generally be ascribed to an

infarct from a valvular vegetation, and infarcts in any other organ may be assumed to arise from the same cause.

§ 199. **Symptoms in pericarditis.**—Until the introduction of auscultation, dry pericarditis was a disease only discovered on the post-mortem table. The only evidence we have of its presence is the characteristic superficial to-and-fro murmur produced by the movements of the heart. Its discovery is usually accidental, and made when the heart is examined as a matter of routine. There is no other distinctive sign associated with it, and, in marked contrast to dry pleurisy, it is essentially a painless complaint. When pains are associated with its presence it will invariably be found that there is evidence of a myocardial affection. This curious painlessness of pericarditis compared with pleurisy is one that has long puzzled me, and I have only a dim perception of how it may arise. I merely call attention to this fact in passing.

Pericarditis may arise in the course of a number of chronic complaints, as in diabetes and Bright's disease, or in the course of an acute disease, as pneumonia or rheumatic fever. Sometimes one comes across it quite accidentally—the patient, not feeling quite well, consults his doctor, and in the course of an examination this is detected. Such patients may go quietly about their occupation for many weeks with a well-marked to-and-fro murmur, and suffer no further trouble.

When effusion takes place into the pericardial sac there is an increase in the area of the cardiac dullness, which assumes a somewhat characteristic shape. It reaches up to or above the second rib, and if the area be mapped out it will have a somewhat pear-shaped character. There is an absence of the heart's movements at the lower point to the left, and this should always arouse the suspicion of effusion where there is an increase in the heart's dullness. Ewart describes a small area of dullness behind at the base of the left lung. This is important to remember, for an increase in the size of this area may cause an extensive pericardial effusion to simulate fluid in the pleural cavity. I have tapped a pericardial purulent effusion in mistake for an empyema, and my mistake arose from not ascertaining the position of the heart's movements. Had the case been one of pleural effusion, I should have found the heart beating to the right of the sternum; but the whole left chest was dull, so that the idea of it being pericardial never crossed my mind.

The question of pericardial effusion embarrassing the work of the heart has arisen on account of the results of experimentally distending the sac with fluid. I have never found any very serious embarrassment of the heart from extensive pericardial effusion, the reason being probably that while the normal pericardium is a more or less inelastic bag, with the

inflammatory invasion it becomes distensile, and therefore able to accommodate an enormous amount of fluid with little embarrassment to the heart.

§ **200. The heart in rheumatic fever.**—The real nature of the changes that take place in acute affections of the heart are being gradually revealed, and it is now possible to connect many of the obscure signs observed during life with the disease process in the heart. Many workers have contributed to our present knowledge, but the following description is taken more particularly from the observations of Cowan [385] and of Poynton and Paine [422], as they have sought with some degree of success to correlate their pathological findings with clinical and experimental data. These observations have been worked out more particularly in rheumatic affections of the heart; but similar changes have been found in other acute affections, as in pneumonia, diphtheria, influenza, septic poisoning.

According to Poynton and Paine, the heart trouble starts with the invasion of the heart by the specific organism of rheumatic fever—the Diplococcus rheumaticus. (Many later investigators have failed to isolate this organism.) They reckon to have isolated this organism from vegetations found on the valves of the heart and pericardium in acute rheumatism, have cultivated it, and produced changes in animals identical with rheumatic endocarditis, myocarditis, and pericarditis. The invasion of the endocardium usually affects first the base of the valves, and produces swelling and infiltration of the margins. The swollen edges may break down and ulcerate, or vegetations may form. The course of the disease varies greatly, from the simple endocarditis which recovers, to extensive ulceration of the valves, associated with the severe symptoms characteristic of malignant endocarditis.

The myocardium rarely escapes, and the changes in it are of great importance, both for the acute condition and for the subsequent integrity of the heart muscle. Fatty degeneration and breaking down of the muscle-fibres are fairly common, while the specific organism has been found accompanied by cellular infiltration. There may be congestion of the blood-vessels, exudation of the leucocytes, and swelling of the connective tissues. Aschoff [256] describes the occurrence of numerous cellular foci.

During the acute attack extreme dilatation may occur, due probably to a toxaemic poisoning of the heart muscle, as no microscopic change may be present. This poisoning is probably also the cause of the extreme weakness and irritability of the heart, which persists for some time after the subsidence of the fever. The pericardium is also liable to invasion, and here the changes may vary from a slight transient pericarditis to an extreme inflammation, which does not entirely subside, but lingers on, forming adhesions to the tissue outside the pericardium and penetrating to the heart itself.

A process of slow cicatrization often follows, producing changes in the valves, heart muscle, and pericardium that seriously embarrass the heart in its work in after years.

Symptoms.—Attacks of rheumatic fever may complete their course with no affection of the heart. In some instances the heart may be affected and give rise to no positive sign, and it may be only after months or years that a murmur or irregular action indicates that there must have been some affection of the heart which the symptoms of the subsequent sclerosing process has revealed.

Generally, however, we can recognize certain changes in the heart's condition, chiefly in an increase in the size and the presence of a murmur. These cardiac changes may go on with very little increase of temperature and little or no evidence of joint trouble. Sometimes in these milder cases I have detected evidence of involvement of the a.-v. bundle by signs of interference with its power of conveying the stimulus from auricle to ventricle.

With more serious involvement the dilatation of the heart may be extreme, and Lees and Poynton [110] particularly have called attention to the enlargement, and the ease with which it may be mistaken for pericardial effusion. At first, the full extent of the enlargement may not be realized, because it is partly masked by the lung. When the lung is pushed aside, the greatly enlarged heart can then be readily recognized. The rate of the heart is usually greatly increased beyond what might be expected from a mere rise in temperature. The pulse becomes soft and compressible, and sometimes shows irregularities whose nature in all cases I have not been able to make out. With subsidence of the fever, the patient enters on a long and slow convalescence. Other cases do not terminate so favourably, especially if the heart has been damaged by a previous attack. Complications, as pneumonia, are apt to arise. In severe cases there may be a considerable amount of praecordial distress. The breathing becomes shallow and rapid. The patient feels easiest with his shoulders well raised. The face becomes dusky, the lips dark red, sleep is broken and fitful, and the patient is continuously altering his position. The mind wanders and mental delusions arise.

From such a state as this the young during their first attack may recover, but in the middle-aged the condition is very serious. Attacks of syncope may appear, and the patient may die in one. Frequently they gradually sink in spite of all treatment and die.

In the recurring attacks of rheumatic fever this question of previous damage to the heart is a very important one. Patients with damaged aortic and mitral valves may pass scatheless through serious attacks of

rheumatic fever, presumably where the heart is not involved in these later attacks. When, however, the process lays hold on the heart, the patient's life is in great danger, and after a period of extreme suffering the struggle frequently ends in death.

While the foregoing description gives briefly the main points of the heart affection in rheumatic fever, it also holds good for the condition in other infectious diseases, apart from the recurrent attacks. As, however, the presence of other lesions has a modifying effect upon the course of the disease, it is necessary to refer to them. Unfortunately, the reference can only be brief and in the main unsatisfactory, the analysis of the symptoms in these cases having been very imperfectly carried out.

§ 201. **The heart in pneumonia.**—The invading organism may assail the heart as well as the lungs, and the course of the illness be rapid and severe. It is difficult to distinguish between the changes due to invasion

Fig. 140. Febrile pulse of low arterial pressure, T. 103°, P. 116, R. 36. This was taken eight hours after the rigor at the beginning of a pneumonia. This and the four following tracings show a type of asthenic pulse.

of the heart from those due to the general infection. In severe cases the evidence of the heart affection is very prominent. Within a few hours of the initial rigor, and before there is any pulmonary sign, the evidence of the heart affection is all too apparent. The patient may be young and, prior to the attack, a perfect specimen of youthful health and vigour. A few hours after the rigor, the temperature may be over 102° F. The condition of the pulse is the best guide to the state of the heart at this stage, and shows ominous signs of what is to follow. It is greatly increased in rate, 115–130 per minute. It is soft and compressible, and offers no resistance between the beats. The peculiar manner in which it impinges against the finger—sharp and short, then quickly subsiding, indicating an absence of sustained pressure—is always to me a serious sign. It is usually associated with greatly relaxed arteries, and a sphygmogram shows little or no sign of a dicrotic wave (Figs. 140–4), indicating great lowering of the pressure during the diastole of the heart. The heart itself shows little definite sign. The sounds are short and sharp at first, becoming later somewhat muffled. A certain amount of dilatation occurs, detected more particularly to the right of the sternum. Usually in these cases the end comes with tragic suddenness; the rate of the pulse increases, irregularities appear, and the patient succumbs

within three or four days of the initial rigor. Figs. 140–4 show the characteristic features of the pulse in acute fatal pneumonia in a young previously strong and healthy adult.

There are two conditions I have come to look upon as signs of grave complications in pneumonia—the occurrence of an occasional irregularity before the crisis, and a pulse-rate over 140 per minute. Neither of these is

FIG. 141. T. 101·5°, P. 96, R. 28—second day.

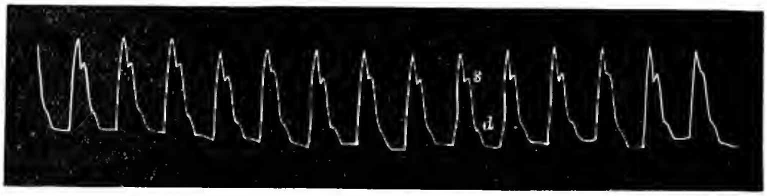

FIG. 142. Asthenic type of pulse with well-marked systolic wave *s*, and only a faint indication of the dicrotic wave *d*—third day.

FIG. 143. T. 103°, P. 124, R. 48—fourth day.

FIG. 144. T. 102°, P. 148, R. 52. The irregularity and rapidity of the pulse heralded the fatal end on the fifth day.

necessarily a fatal symptom. I had pointed this out in my book on the pulse, and John Hay[402], analysing 200 cases of pneumonia, found a small number recover who had shown an irregularity preceding the crisis. I therefore went into the matter more carefully, and found that the occasional irregularity might be due to more conditions than one, and in all my fatal cases the irregularity was due to exhaustion of the contractility, as shown in Figs. 137 and 138; but this subject needs further elucidation.

§ **202. The heart in diphtheria.**—The complications here are so

varied that danger may arise in several quarters. The heart muscle itself may be the seat of profound changes, the symptoms somewhat resembling those in rheumatic fever. But in diphtheria more than in any other acute disease there is a tendency to fatal syncope, and I do not understand how this is brought about.

§ **203. Septic infections.**—There are a great many septic infections that injure the heart either from toxaemia or by a specific organism invading the heart. In the latter case the endocardium is frequently attacked, and the disease is then described as septic endocarditis. In these cases there is also invasion of the myocardium, and it is the profound depression of the heart muscle which is often the grave element.

In an account of 150 cases of infective endocarditis, Horder[405] states that in 90 per cent. of cases a culture of a pathogenic organism can be obtained. The following table gives the analysis of forty positive results of blood culture during life :—

Number of Cases.	Micro-organism isolated.
26	Streptococcus
5	Bacillus influenzae
5	Pneumococcus
2	Gonococcus
1	Staphylococcus albus
1	Unclassified.

The illness in these cases usually begins insidiously, and at first may be mistaken for some trivial febrile complaint, or for influenza. Soon, however, the extreme prostration of the patient, the recurrence of rigors, and the patient's own sensation of illness, show that the condition is of a more serious nature. Usually also there is excessive perspiration. If the heart be watched it will be found to dilate and a systolic murmur appear. The true nature of the condition may not be revealed until the detachment of a vegetation produces hemiplegia, or an infarct in the spleen or kidney or elsewhere, and death may speedily supervene (malignant endocarditis).

Other cases may linger on with indefinite febrile attacks, the patient sick, pale, and ill, and the real nature of the illness be a mystery. In some of these cases very little change takes place in the heart. I have seen a case after confinement have slight fever for nine weeks with no change in the size of the heart, the rate generally about eighty per minute, a rough systolic mitral murmur the only abnormal sign, until an attack of hemiplegia led to the recognition that the rough mitral murmur was due to vegetations on the valves. Osler[418] has recently published an account of ten cases of chronic

infectious endocarditis. In addition to the irregular fever he gives the following as the most suggestive features which help to identify the nature of the disease. (1) A knowledge of the existence of an old valve trouble; (2) the occurrence of embolic features, sudden swelling of the spleen, sudden attack of haematuria, embolism of the retinal arteries, hemiplegia, or the blocking of a vessel in one of the limbs; (3) the onset of special skin symptoms, purpura, and more particularly the painful erythematous nodules, in all probability due to minute emboli; (4) the progressive cardiac changes, the gradual increase in the dilatation of the heart, the marked change in the character of the mitral murmur, the onset of a loud rasping tricuspid murmur, or the development under observation of an aortic diastolic bruit.

In pyaemia and puerperal septicaemia we get conditions of profound gravity caused by certain organisms. The pulse in these cases gives the most trustworthy information. It is small, soft, and easily compressed, not necessarily very rapid, and the temperature need not be high (101–2°). The heart shows little change except that the sounds are feeble, the patient is lethargic, the face is slightly sallow or pale and sunken. The aspect of the patient, taken with the rate of the pulse, affords assistance in recognizing the condition. I dwell upon this because, happily, some of the younger members of the profession have little experience of dangerous forms of puerperal fever, but, having heard of the terrors surrounding them, are not infrequently unnecessarily frightened at the occurrence of a post-partum rise of temperature of trivial significance, while others do not recognize the significance of the heart symptoms when danger actually arises.

§ **204. Treatment.**—When the real nature of the trouble is appreciated in acute affections of the heart, it will be realized how powerless we are directly to modify the diseased process. In vaccine or serum therapy there is a promise of a specific remedy in each case to meet the special organism causing the mischief. But so far our efforts have been attended with little success. Horder [405] states that of thirty-nine cases treated in this way only one recovered, and unfortunately in this case no micro-organism was demonstrated in the blood. He says, ‘ I have given the treatment most thorough trial in several cases, and occasionally seen temporary improvement result, but never any permanent good. Conder [381] has recently described a case where the recovery seemed directly due to the vaccine injection.

In rheumatic fever the salicylates seem to have an undoubted action on the course of the disease, and it may be that the drug can modify the heart affection. Their employment is so frequently of use that it should always be tried, even pushing it at times as Lees advocates. I have not given the large doses of the salicylates recommended by Lees in a sufficient number

of cases to be able to dogmatize, but as the few in which I have followed his directions seemed to get benefit, I have included in Chapter XXXIII a short account by Dr. Lees of the method he pursues.

Apart from the probably specific action of salicylate in rheumatic cases, the employment of cardiac or other drugs is of little avail.* The heart is already in possession of a poison far more powerful than the drugs at our command, and these in medicinal doses are without effect. But on this account it must not be supposed that all treatment is useless ; rather should it direct our attention to the consideration of other means of treatment. The man who puts his faith in drugs exclusively neglects too often the most useful methods. Recognizing that the heart muscle is greatly embarrassed in its work, the endeavour should be made to give it as little work to do as possible, and to save it from all sources of irritation ; in fact, to place it in a condition of rest, as far as rest is possible for such an active organ as the heart. To this end the general condition of the patient should be carefully studied, the position he assumes should be one that gives the heart least work to do, the food should be so administered that while nourishing him it does not lead to abdominal distension, his bowels should be so regulated as to act freely without straining. As fidgeting and restlessness keep the heart irritable and variable in its action, everything should be done for his bodily comfort—sponging, and arranging his pillows and the bedclothes, and the many little things that a deft and intelligent nurse can suggest. Above all, sleeplessness, which is so often present, or the sleep that is broken and disturbed, should have most careful consideration, and suitable hypnotics be given as described in Chapter XXXIII.

When there is reason to suspect that the heart muscle has been affected by the illness, great care should be taken during the convalescence to give the heart muscle time to recover. Any cause, effort or excitement, that accelerates the heart's action, should be avoided, and exertion only permitted when the dilatation has subsided, and effort ceases to call forth any disagreeable sensation. It may be weeks or months after the fever before the heart muscle recovers.

* Horder says: '"Blood antiseptics" seem doomed to failure in dealing with pyogenic blood infections, because it is not possible to get the drugs into the blood in a nascent or active condition. Combination with the proteids of the blood-cells or the plasma takes place before the drug comes into contact with the micro-organism. Quinine, mercury, arsenic, carbolic acid, formalin, and many other reputed remedies, all fail, whether administered by the mouth, subcutaneously, or intravenously. The sulpho-carbolates which have had a special vogue are equally disappointing ; I have used them in very large doses in several cases without any results. Silver salts in combination with nuclein have been highly spoken of in the treatment of septicaemia, but here again I have never seen any good results in infective endocarditis. The same remarks apply to yeast and its active principle.'

CHAPTER XXV

Valvular Defects

§ **205. The manner of heart failure with valvular defects.**—It is manifest that valvular defects can embarrass the work of the heart in two ways: first, by narrowing the orifice and thereby impeding the outflow; second, by imperfect closure so that leakage occurs.

The defects are recognized clinically mainly by the presence of murmurs, but it must not be assumed that absence of murmurs implies an intact valvular apparatus, for great widening of an orifice and large regurgitation may take place when no murmur can be detected. As valvular lesions are produced by a variety of conditions, it might have been more logical to discuss them under the heading of these conditions. As, however, they are presented to us at a stage when all immediate symptoms of their causal condition are in abeyance, it is more convenient to describe them at the time that the heart-changes evoke symptoms of exhaustion. Years may elapse after the mischief has been done to the valves before symptoms arise that call attention to the heart trouble. In the acute condition producing the valvular lesion, the matter is presented in an entirely different aspect, for the febrile state and the symptoms associated with the cause of the fever predominate. In chronic valvular affection the symptoms only arise where exhaustion of the heart muscle sets in. The symptoms due to exhaustion appear at varying periods after the damage has been done to the valves, and the time of the appearance of these symptoms depends on the degree of embarrassment offered to the heart's work by the damaged valve, on the

condition of the muscle-wall, and on such accessory factors as tend to exhaustion, as over-exertion, excessive food, drink, and so forth.

In organic lesions of the valves it must always be borne in mind that the sclerotic process causing the lesions may be progressive, and that there may also be present advancing changes in the heart muscle.

Generally speaking, the symptoms of heart failure show little that is distinctive of the particular valves affected. In the aortic cases the reflex sensory phenomena are more prominent, and the ashen colour of the face is sometimes characteristic. Where there is a mitral lesion, the pulmonary symptoms are usually more prominent, and the face may be ruddy with a dark tinge. Apart from such differences there is a great similarity in the symptoms of heart failure produced by all kinds of lesions.

Mitral Stenosis

§ 206. Conditions inducing heart failure in mitral stenosis.— This is perhaps the most common of valvular defects with which heart failure is associated. It arises generally in consequence of rheumatic endocarditis, though it may be found in people with no rheumatic history, and a previous history of erysipelas, or some other febrile complaint, may give a possible clue to its origin.

The condition is never recognized during the acute process which induces it, for the reason that its presence can only be detected when the cicatrizing process following the inflammation narrows the orifice, and on account of its origin in scar-formation it is often a progressive lesion. Once the stenosis is present it may remain moderate in amount, and offer so little embarrassment to the heart that patients may reach extreme old age with no heart failure. As a rule, however, the cicatrizing process goes on with varying rapidity until in some cases the mitral orifice is reduced to a mere slit, and the valves resemble a thickened calcareous diaphragm. It is important to bear in mind the progressive nature of the lesion, for it accounts for the varying changes in the symptoms. It should also be borne in mind that the cicatrizing process may be going on in the muscle, causing contraction of the chordae tendineae, impairing at other places the functional activity of the heart muscle, and affecting the a.-v. bundle, depressing the conductivity or producing the nodal rhythm, thereby profoundly modifying the nature of the rhythm of the heart.

From this it can readily be understood that the manner in which heart failure is brought about in many cases is somewhat complicated. In some, embarrassment may not ensue until the narrowing of the orifice has become extreme. In others there may be a fatal issue while the narrowing is yet

moderate. In the latter cases the muscle-wall will inevitably be found to have been damaged. In a third class of case the cicatrizing process has extended to the a.-v. bundle, and encroaching on it has induced the nodal rhythm, characterized sometimes by greatly increased frequency of the heart's action, and usually by a continuous irregularity (see Chapter XX). This irregular action further embarrasses the heart muscle in carrying on its work, as already described. The mechanical embarrassment may not only lead to increasing back-pressure involving in succession the left auricle, pulmonary circulation, and right heart, but the small quantity of blood passing into the left ventricle leads to the tissues generally being poorly nourished. The left ventricle, suffering likewise from an impoverished supply of blood, may offer independent symptoms of exhaustion, as for instance by dilating (exhaustion of tonicity), and possibly by reflex phenomena, as in the rare instance of an attack of angina pectoris in mitral stenosis.

§ 207. **The murmurs present in mitral stenosis.** *The presystolic murmur.*—The presystolic murmur—the auricular systolic murmur of Gairdner[396]—is due to the contraction of the left auricle forcing blood through the narrowed mitral orifice. With the varying changes in advancing cicatrization the murmurs of mitral stenosis alter, and present peculiarities that have hitherto not been sufficiently appreciated. In the very early stages,—some years before the appearance of a murmur,—I have detected a slight presystolic thrill. The first murmur to appear precedes or runs up to and seems to terminate in the first sound, and is audible over a small area around the apex. This murmur may vary in duration, being usually short and abrupt, but it sometimes begins earlier and is somewhat prolonged. It is of a crescendo character, rising in pitch till it ends in the first sound. Now although this is the usual position in the cardiac cycle of the presystolic murmur, I have found a few cases in which it did not terminate abruptly in the first sound, but was separated from it by a very brief interval. In some of my cases I asked a number of my colleagues to mark out on a tracing of the radial pulse the exact position of the murmur in the cardiac cycle, and every one without hesitation indicated the position as represented in Fig. 112, where the loudest part of the murmur is separated by a minute interval from the first sound. When a jugular or an apex tracing was taken it was seen that the position was identical with that of the auricular systole. In other words, there was a delay in the transmission of the stimulus for contraction between auricle and ventricle. This delay can sometimes be increased by digitalis, and the position of this murmur in its relation to the first sound moves in the same way.

I mention this not only to enforce the evidence of changes in the a.-v.

bundle in mitral stenosis, but because some clinicians deny that the auricular systole causes the presystolic murmur in mitral stenosis. I have long been puzzled to account for their denial of what seems so obvious, but I have found what seems to be the explanation, and will deal with it after describing the murmurs with the nodal rhythm.

The diastolic murmur.—With advancing stenosis of the orifice another murmur makes its appearance, namely, one occurring immediately after the second sound, heard only in the immediate neighbourhood of the apex beat. At first it is very faint, and not very constant, but it usually increases in duration until the whole diastolic period may be filled up by it. This diastolic mitral murmur diminishes in intensity from the beginning—differing thus in its diminuendo character from that of the presystolic. Frequently we can detect a continuous murmur during the diastole of the heart, beginning loudly, falling away, then increasing in intensity. The first or diminuendo portion of such a murmur is the diastolic mitral murmur, while the terminal crescendo portion is the presystolic. The cause of the diminuendo diastolic mitral murmur is the flow of the blood that has been accumulated in the auricle during the ventricular systole through the narrowed mitral orifice ; this begins as soon as the mitral valves open, that is, when the pressure in the ventricle falls below that in the auricle.

The disappearance of the presystolic murmur.—The next change in the character of these murmurs is the sudden disappearance of the presystolic crescendo murmur, while the diastolic murmur persists. Usually this change occurs with the onset of grave symptoms of heart failure, the heart's action becoming rapid and irregular. At other times the change takes place with no serious symptom, but the heart invariably becomes irregular. I have explained this fully in Chapter XX and in the Appendix (II), as being due to the fact that the rhythm of the heart starts no longer at the normal place but lower down, perhaps at the a.-v. node, and that in consequence the auricular contraction no longer precedes the ventricular, but the auricles and ventricles contract together. Clinicians hitherto have not recognized the fact that in mitral stenosis the presystolic murmur disappears with the onset of the continuous irregularity of the heart.

When the heart's action is slow there is no difficulty in recognizing the diastolic murmur, and the absence of the presystolic. The diastolic murmur is sometimes of great length, starting immediately after the first sound, and slowly dying away, as in Fig. 57. When the heart's action is rapid, this diastolic murmur may fill up the whole diastolic pause, and it might hastily be assumed that the murmur was presystolic. But if it be carefully auscultated, it will be found that it is not crescendo in character ; when a longer

pause occurs it will be found that the murmur stops short before the first sound, so that there is a silence between the end of the murmur and the first sound (see shading in Fig. 57). In these cases the jugular and liver pulses are invariably of the ventricular form.

The presystolic murmur of ventricular origin.—In the careful examination of a large number of these cases of nodal rhythm we find that the first sound has not the sonorous rumble of the normal first sound, but is represented by a sharp, short snap of very brief duration. Preceding this snap, in a very few cases I have heard a brief crescendo murmur, but a venous tracing showed that there was no auricular systole at the normal time. From this I suggest that the disputants in the matter have hitherto been confusing two separate conditions. As I have already pointed out, the evidences of auricular systole producing a presystolic murmur are indisputable. While the auricular systole is one cause, it is obvious there must be another cause in these cases of nodal rhythm, and I therefore accept the view that a slight regurgitation through the mitral orifice at the beginning of ventricular systole may produce the brief presystolic murmur. (As a matter of fact such a murmur does not precede the ventricular systole, but only precedes that portion of the first sound represented by the terminal snap).

Systolic murmur due to mitral stenosis.—There is another form of murmur associated with mitral stenosis, systolic in time, and heard best at the apex. It is peculiar in the respect that it begins a minute interval after the first sound, and rises in intensity until it terminates in the second—a systolic crescendo murmur, in marked contrast to the usual form of mitral systolic murmur. I have watched for years a number of cases who had this peculiar murmur, and have noted that the majority had a history of rheumatism in their youth, but I had no case that came to a post-mortem examination. In conversation with Dr. David Drummond, he told me he was acquainted with the murmur, and that it was usually associated with mitral stenosis.

All the murmurs due to mitral stenosis have usually a very limited area of propagation, being heard over a small space immediately surrounding the apex beat. Occasionally we meet with presystolic murmurs heard over the whole heart.

§ 208. Progress and symptoms in mitral stenosis.—From the progressive nature of the lesions in the valves and in the heart muscle, it will be realized that the symptoms are not constant.

The patient comes first into consideration mostly in early or middle adult life. The complaints then are shortness of breath, a sense of suffocation, and palpitation on exertion. In some the face is ruddy, with a hue

a shade darker than is compatible with the ruddy countenance of robust health. At this stage there is little or no increase in the size of the heart and no dropsy. A presystolic murmur can usually be detected. The patient's complaints may be the only evidence we have of the heart failure, and these point to an exhaustion of the reserve contractile force. After a period of rest this exhaustion may disappear and the patient may go on for years with but little further trouble. After a time, however, some again break down, and the symptoms complained of may be of the same nature. Frequently, however, a change is found in the character of the murmurs—a diastolic murmur usually being perceived, and there is sometimes a longer duration of the thrill, these signs implying an increased narrowing of the orifice. On the other hand, in those in whom no further narrowing takes place, the murmur does not change, and the patient may go on for many years, and, if a female, may bear children, with no breakdown. In these cases we can infer that there is no progressive muscular or valvular sclerosis. With the increased narrowing of the orifice, as indicated by the appearance of the diastolic mitral murmur, the heart becomes much embarrassed, the symptoms become much more distressing, and finally dilatation of the heart (failure of tonicity) may set in. But even without the progressive narrowing, dilatation may appear early, and then it may be inferred with certainty that the rheumatic process has permanently injured the heart muscle.

The rhythm of the heart may become continuously irregular, from the cicatrizing process affecting the a.-v. bundle, and with the onset of this nodal rhythm further embarrassment arises as described in Chapter XX. If there be no change in the size of the heart with this nodal rhythm, and no great acceleration of rate, the heart failure may be very slight in degree, but if the heart dilates, especially if the rate is accelerated, then all the extreme symptoms of heart failure follow (dropsy, enlargement of the liver, &c. See Chapter XX).

In the vast majority of cases the heart recovers from its first breakdown and usually from many subsequent attacks. Indeed, after one attack, I have known patients go on for twenty years and more with no further trouble beyond a slight limitation of the field of response to effort.

After repeated attacks, the patient's life becomes one of great limitation. The future depends often on the rapidity of the advance of the sclerotic process of the valves and the heart muscle. If the rate of advance be slow, and the heart muscle capable of responding to treatment, the patient may go on for many years with a crippled existence. Sometimes we find in the young, about twenty years of age, at the post-mortem examination, the

orifice narrowed to a mere slit. In others we find the mitral orifice not much contracted but the heart-wall greatly dilated, and evidence of fibrosis of the muscle. Hence it will be seen that the progress of these cases is largely dependent on the rate of change in the muscle as well as in the valve. The final issue is usually by great extension of the dropsy and exhaustion.

There are several complications which may arise.

§ 209. **Occasional symptoms.** *Paroxysmal tachycardia.*—In place of the nodal rhythm being permanently established, it may appear intermittently as attacks of paroxysmal tachycardia. These attacks are of varying importance. Some patients may have them for more than twenty years and seem little the worse. Others may have them occasionally, and then the heart settles down with the rhythm permanently altered. In such cases the future depends on whether the heart slows down or remains at a greatly increased rate, as already described (§ 160, also Case 11, Appendix II).

Haemoptysis.—At various stages patients may be seized with great bleeding from the lungs. Here doubtless the cause is the back-pressure in the pulmonary circulation and rupture of the blood-vessels. As a rule this is a grave sign, the patient dying sometimes shortly after an attack.

Cerebral embolism.—Vegetations may exist at the mitral valves, with no certain sign of their presence until a small portion is detached and impacted in some vessel, giving rise to a hemiplegic attack, or an attack of aphasia. Usually recovery takes place speedily, and may be permanent, but cases have been recorded in which the aphasia or hemiplegia has remained complete for many years.

Attacks of angina pectoris.—Although very rare, these may occur in mitral stenosis. In the few cases I have observed they were all secondary to some excessive exertion, and the patients had only one or two attacks, remaining perfectly free from them for years afterwards.

Mitral Regurgitation

Mitral regurgitation may be the result of a damaged valve or of dilatation of the orifice from depression of the tonicity of the muscles supporting the valves.

§ 210. **Murmurs due to mitral regurgitation.**—The murmur of mitral regurgitation is systolic in time, heard loudest at the apex. It may be soft and blowing, of little intensity, and heard over a very limited area, or propagated into the axilla. Or it may be rough and loud, and heard over the whole heart and round to the back of the chest. It is not always possible to tell whether it is due to dilatation of the orifice or to damage

of the valves. The rough loud murmur with an accompanying thrill is always a sign of damaged valves.

§ 211. Conditions inducing heart failure in mitral regurgitation. —When the muscle is unimpaired, little or no bad effect may follow damage to the mitral valves. Even where the regurgitation is due to 'functional' dilatation of the orifice from depressed tonicity, the contractile power of the muscle may maintain a good and efficient circulation. The really serious trouble in connexion with mitral regurgitation arises when the muscle is impaired and the regurgitation is due to a complication of the dilated orifice and diseased valve. The subsequent results depend on the degree of the exhaustion of the muscle of the heart. The backward pressure resulting from the regurgitation embarrasses the left auricle, pulmonary circulation, and right heart. The degree to which this may extend depends in a great measure on the tone of the heart muscle. While back-pressure is a factor of importance and may be a predisposing cause, yet it produces comparatively few symptoms until the tonicity gives way, which is manifested by dilatation of the heart. The dilatation is generally looked upon as the result of the regurgitation, the back-pressure ultimately producing yielding of the walls of the right heart. This is not quite correct, for long before there is any back-pressure we may find evidence of a dilated right heart. If we examine carefully the condition of the heart when the valves have been damaged by rheumatic endocarditis, during one of the slight attacks of heart failure which are liable to occur after over-exertion, we may find the heart slightly dilated, the right ventricle being in front so that the left ventricle is pushed to the left behind the lung; the apex beat is then due to the right ventricle, which gives a negative cardiogram (Fig. 33). After a few days' rest and treatment, the right heart may retreat and the apex beat is then due to the left ventricle, the cardiogram now presents the normal characters, rising during systole. In such cases there is no evidence whatever of pulmonary engorgement and back-pressure. In fact, in the majority of cases, as Graham Steell[180] says, the change in the 'valves is altogether inadequate to explain the evidently free regurgitation that occurred during life, and the disastrous dilatation of the heart. The muscle-failure factor, it may be presumed, was the essential one'.

The damage to the valves is most commonly the result of rheumatic endocarditis, and, as we have seen, the process is rarely limited to the endocardium, but invades the myocardium. Septic endocarditis may also damage the valves. In all cases of mitral stenosis there is mitral regurgitation, but the amount of the regurgitation is never so marked as to be the serious factor in the case.

Serious regurgitation occurs through the mitral orifice with the valves uninjured in the latter stages of many affections, but more particularly in renal disease and cardio-sclerosis. Here the condition is brought about by the failure of the muscle to support the orifice, and this is too often the sign of a final and fatal exhaustion of the heart muscle (see Chapter XXVII).

It will thus be seen that the symptoms produced by mitral incompetence are only of gravity when there is also muscle failure, and this is dealt with in sufficient detail in the chapter on Dilatation of the Heart (Chapter XXIII).

CHAPTER XXVI

VALVULAR DEFECTS (*continued*)

AFFECTIONS OF THE TRICUSPID VALVES

LESIONS of the tricuspid valves are rare, and are nearly always associated with similar lesions in the mitral and aortic valves. The heart failure associated with these lesions is never due to the tricuspid lesion alone.

§ **212. Tricuspid incompetence.**—Although actual disease of the valves is rare, incompetence of the tricuspid orifice is extremely common—so common, indeed, that I am inclined to look upon the valves as being barely able to close the orifice perfectly. This view is based upon the observation of many patients, in whom I have been able to detect a tricuspid systolic murmur with no appreciable increase in the size of the heart. The murmur in many cases is very fugitive, being present in the first few minutes of an examination, and disappearing when the heart becomes quieter. A consideration of the size of the orifice and the size of the valves led John Hunter [338] to doubt their competency, while Mayo [341] declared that the tricuspid valves never perfectly close the orifice. Experimentally it has been found impossible to raise the pressure in the right ventricle, on account of the ease with which regurgitation takes place through the tricuspid orifice.

The slighter forms of tricuspid murmurs are limited to a small area over the middle of the sternum. With increase in the size of the right heart they may be heard over the whole anterior surface of the heart. They are often associated with mitral systolic murmurs, but one can usually detect a difference in quality in the mitral murmur heard beyond the left nipple line and in the axilla from the tricuspid murmur heard over the middle of the sternum.

It should never be concluded that no tricuspid regurgitation occurs

because of the absence of a murmur, for it is of frequent occurrence to find evidence of tricuspid incompetence in the character of the jugular and liver pulsation (ventricular form), and in the greatly widened orifice post mortem, while during life there was no systolic tricuspid murmur. A weak muscular wall and a wide orifice may give rise to no murmur.

I dwell at some length on these points, not because the tricuspid regurgitation is of much practical value, but because misunderstanding of its symptoms has led to a wrong construction being put on the effects of tricuspid regurgitation, and to the real significance of the ventricular form of the venous and liver pulse being missed. I have already pointed out that slight regurgitation in a normal heart would add to the accumulating blood in the right auricle during ventricular systole, and would therefore be a factor in the production of wave *v* in the jugular pulse. Now most writers overlook the fact that a dilating auricle is interposed between ventricle and jugular, and have assumed that as soon as tricuspid regurgitation takes place a wave appears in the jugular at the beginning of ventricular systole. They have therefore regarded the ventricular form of the venous pulse as only a sign of tricuspid regurgitation, and have missed the real significance of this very important symptom. That it is a sign of tricuspid regurgitation there is no doubt, but it is a sign of far greater significance, namely, that the auricle does not precede the ventricle in the cardiac cycle. This is illustrated in Chapter XX and Appendix II.

§ 213. **Tricuspid stenosis.**—In the majority of cases tricuspid stenosis is not recognized during life, as the symptoms produced are not always distinctive. It is only rarely that a presystolic tricuspid murmur is heard; I have only heard it in three cases, in which it was present in a very limited area over the middle of the sternum. There is usually present also a mitral presystolic murmur at the apex, but as each murmur is confined to such limited regions I have had no difficulty in distinguishing them. In one case the auricle had become so greatly hypertrophied that it sent back a large wave into the jugular, and that with such force that it caused the valves in the jugular and subclavian veins to close with a snap, which I could hear over these veins as a clear, sharp sound preceding the first sound.

As a result of the stenosis of the tricuspid orifice, the right auricle hypertrophies, and on this account sends a wave back into the vein with such force that it distends the liver, and I therefore look upon pulsation of the liver with a marked wave due to the auricle as an evidence of possible tricuspid stenosis (§ 120).

§ 214. **Disease of the aortic valves. Etiology.**—By far the greater

number of cases of affection of the aortic valves owe the lesions primarily to one of two conditions—rheumatic endocarditis and the sclerotic process accompanying arterial degeneration. Under rare circumstances the valves may rupture, but here there is usually some antecedent disease of the valve. Congenital defects may in rare cases give rise to great embarrassment of the heart.

It is the lesions induced by the two first-named conditions that require most consideration. In both instances the condition is usually well established before it is found out. In many cases the presence of aortic changes is discovered accidentally, when a systematic examination is being made for other ailments, or for insurance or a health certificate.

The heart failure in aortic valvular disease is rarely due to this lesion alone. In the majority of cases changes impairing the power of the muscle have been proceeding at the same time as those that induced the valvular changes.

In the rheumatic cases there is frequently present a complicating lesion of the mitral valve.

When the valvular disease embarrasses the heart's work by the extent of the lesion, as by great incompetence, and the heart muscle is healthy, the latter responds to the obstruction to its work by hypertrophy, and this may proceed to an enormous extent, giving rise to one of the largest of human hearts—the Cor Bovinum.

§ **215. Aortic stenosis.**—Aortic stenosis is often associated with aortic regurgitation, and the symptoms of the latter usually dominate the situation. When there is little or no regurgitation, the symptoms of aortic stenosis, being less prominent, are often only detected accidentally in the routine examination of the patient.

The sign most characteristic of aortic stenosis is a murmur systolic in time, heard loudest over the second right costal cartilage, and propagated into the carotids. It may be faint—a mere whiff, or it may be prolonged, and accompanied by a thrill perceptible over the upper part of the chest-wall. The heart's rate is often slow, between fifty and sixty beats per minute. The radial pulse is sometimes very characteristic. It impinges against the finger in a slow, leisurely fashion, and a sphygmographic tracing may show a slanting up-stroke with a slight interruption near the summit (anacrotic pulse, Fig 145), or even a double wave at the top (pulsus bisferiens, Fig. 146). Graham Steell[179] and Lewis[158] state that they have been able to detect this double beat by the finger, and Graham Steell says he has perceived it on one side only. Its real nature is yet obscure.

Beyond these signs there is little that is characteristic in aortic stenosis. There may be symptoms of angina pectoris, but these are due to associated changes in the heart muscle, and other evidences of heart failure can be referred to the same cause.

§ **216. Aortic incompetence.**—The aortic valves being contracted are no longer able to support efficiently the column of arterial blood during the diastole, but permit a backward flow into the heart, and as a result we find certain alterations in the character of the second sound and of the arterial

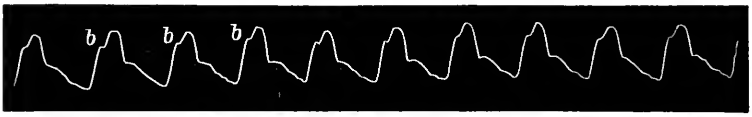

Fig. 145. Anacrotic pulse, from a case of aortic stenosis.

pulse. The closure of the valves no longer gives to the second sound the characteristic snap, but the sound ends in a murmur sometimes long drawn out, sometimes so brief as to be scarcely perceptible—as if the second sound terminated not abruptly but with a faint sigh. The diastolic murmur is usually propagated down the sternum, but sometimes it is heard loudest at the apex. Foster has suggested that this variation in the propagation of the murmur depends on the direction given to the backward flow by the

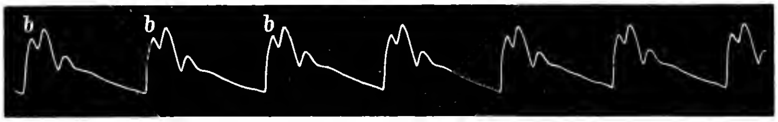

Fig. 146. Pulsus bisferiens, from a case of aortic stenosis.

position of the retracted valve. This seems plausible, but I have not been able to verify it, and the explanation is ignored in recent textbooks.

The regurgitant murmur is usually associated with the murmur of aortic stenosis, and we get the characteristic double aortic murmur (bellows murmur). There is frequently dilatation of the smaller arteries, and this, combined with the effect of the regurgitation on the arterial pulse, causes the artery to become emptier than usual towards the end of diastole. This means a fall of pressure, and in order to maintain a normal mean pressure the heart increases the force of its contractions raising the pressure during systole, so that there is a great increase in the systolic pressure and a great fall during diastole, thus giving rise to the characteristic collapsing pulse (Corrigan's pulse, the water-hammer pulse, Figs. 147, 148, 149). The collapsing character of the radial pulse may be intensified by raising the

arm above the head. At times the arterial pulse is conveyed through the capillaries into the veins, and G. Gibson has obtained a graphic record of such pulsation of the veins on the back of the hand. If the forehead be rubbed so as to produce redness, the flush is seen to wax and wane with each beat of the heart (capillary pulsation).

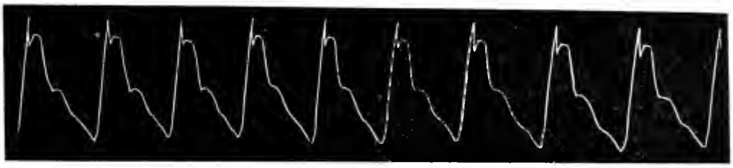

FIG. 147. Pulse of slight aortic regurgitation with good heart muscle.

The double aortic murmur may be detected without any history of a heart affection. In many cases there may be little or no dilatation, and the individual may be able to indulge in games and in occupations requiring considerable exertion with no discomfort. In such cases it may safely be

FIG. 148. Pulse of slight aortic regurgitation with great cardiac failure.

assumed that the damage to the valves has been slight, and that the heart muscle has escaped serious injury.

In other cases the heart is greatly enlarged and the apex beat is diffuse and forcible. The systole and diastole of the heart may cause movements

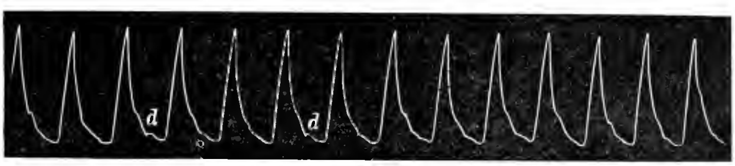

FIG. 149. Pulse of extreme aortic regurgitation with great cardiac failure.

of the liver that simulate pulsation of that organ, but analysis of its graphic records show it to be merely the dragging up and pushing down of the liver by the changes in the size of the heart (Figs. 27, 28). Even under these circumstances the individual may for years pursue an active vocation, but he is always liable to attacks of heart failure. The condition is often associated with affection of the mitral valves, and this is one of the factors

participating in the production of the heart failure that finally terminates these cases.

The most frequent sufferers from aortic valvular disease are the middle-aged. In them the sclerotic process has been gradually advancing, and the early symptoms of exhausted reserve force have been neglected until symptoms of distress command attention. There may have been a history of rheumatism, of excessive drinking, of hard bodily exertion, of syphilis, but, on the other hand, no definite causal condition may be discovered. The facial aspect is frequently pale grey (earthy countenance), though in others it may be full-blooded and ruddy. The complaints are varied. Shortness of breath on exertion, violent throbbing in the neck, attacks of pain over the chest on exertion, are amongst the most common symptoms of which the patient complains in the first instance. For a varying period, under suitable treatment, a certain store of reserve force is gained, and he may go on for months or years, sometimes in fair comfort, but his existence is usually more or less crippled.

The end of these patients is frequently dilatation of the heart, dropsy, and exhaustion. Sometimes this is due to the inception of the nodal rhythm. Those who suffer from angina pectoris may die suddenly. I have seen a few cases die during a sudden attack of dyspnoea of the greatest severity.

§ **217. Prognosis in valvular affections.**—The heart failure depends upon so many and so varied conditions—as the extent of the valvular lesion, its progressive nature depending on the cicatrizing process affecting the valves, the coincident changes in the muscle and in the a.-v. bundle, the conditions of life of the individual—that no rule applicable to all cases can be made. If, however, an attempt be made to appreciate the value of the symptoms present, on the lines I have laid down, an approach to a true prognosis may be made in each case. There is just one point I again wish to insist upon : let no single symptom be the ground for forming a prognosis. In this respect the presence of a murmur has so oppressed the profession that a vast amount of positive harm is continually being done to patients by taking too seriously the prognostic significance of this sign. The field of cardiac response is the only true and safe guide in these cases. Even if for the time being it is limited, judgement should be suspended until an opportunity has been obtained for ascertaining to what extent the heart muscle can regain a store of reserve force (see Chapter XXXII).

§ **218. Treatment.**—As heart failure with valvular defects touches every phase of the subject, the matter of treatment must be discussed from a very wide aspect. The special chapters on treatment therefore include the full consideration of this subject.

CHAPTER XXVII

CARDIO-SCLEROSIS (ARTERIAL DEGENERATION. THE SENILE HEART)

§ 219. Conditions producing cardio-sclerosis. There are certain changes which we recognize as accompanying and giving rise to the features characteristic of advancing years. These changes may be detected in every tissue and organ of the body, and they can be recognized in the bald scalp, white hair, or tortuous artery. The changes in the arteries and capillaries may modify the structure and functions of the various organs, but not all organs equally. Arterial degeneration may in one person be more advanced in the brain, in another in the kidneys, in another in the limbs, in another in the heart. In many these changes are merely those associated with advancing years, and give rise to what we understand by senile changes. When affecting the heart they are usually accompanied by some fibrous or fatty changes in the heart muscle. If these changes are considerable in extent, then we get a train of symptoms which we recognize as due to ‘ cardio-sclerosis ’.

There are two main circumstances that induce degenerative changes (fatty and fibrous) in the heart, namely, the cicatricial changes that follow acute affections, as after rheumatic fever, and the changes that accompany arterial degeneration. Both these conditions affect the muscular structure as well as the valves, and the resulting heart failure is often the outcome of the invasion of both tissues by the sclerotic process. Although it may be convenient to follow conventional lines and describe separately the valvular affections, it must be borne in mind always that in the serious cases there is a widespread condition of which the valvular lesion is but a part.

The changes in cardio-sclerosis due to rheumatic fever have a certain resemblance to those due to arterial degeneration. In both instances there is a replacement of the muscular fibres by fibrous tissue, and a shrinking

of the valvular apparatus, and as a consequence both conditions present identical symptoms. Though there is this resemblance in progress and symptoms, there are other differences which have an important bearing on prognosis and treatment.

The consideration of the rheumatic and other inflammatory forms of sclerosis is included in the chapters on valvular disease. Here I wish to draw particular attention to the changes in the heart that are associated with arterial degeneration and senile changes. The causes of arterial degeneration are still not clear, and it is difficult to say, of the complications in any given case showing arterial degeneration, which are the cause and which the consequence. Clifford Allbutt [58] rightly protests against arterio-sclerosis being considered a disease ; it is the outcome of processes which we imperfectly understand, and may arise as the result of high blood-pressure, toxic conditions, or senile changes. I do not enter into the, at present, hopeless task of distinguishing the causes of the changes in the arterial system. A little of the truth may be present in each of the many competing theories at present holding the field, but no one of them can be considered wholly satisfactory and convincing.

In the meantime, an appreciation of the changes as they affect the heart gives us great assistance in the treatment of our patients.

§ 220. **Conditions inducing degenerative changes in the arterial system.**—It is usual to attribute the changes to some earlier process that has affected the blood or the arteries, of which kidney disease, syphilis, over-exertion, are the most striking examples. But it will be found frequently that extensive arterial degeneration may be present for which one can find no definite cause.

There can be no doubt that affections of the kidneys tend to induce these changes. But in many people the kidney lesions are undoubtedly secondary, and patients may show well-marked and extensive arterial degeneration many years before there is the slightest evidence of kidney ailment. In such instances it is but reasonable to assume that the renal degeneration, like the cardiac and cerebral, is secondary to the arterial degeneration.

§ 221. **Obliteration of the capillaries.**—One of the most striking changes that take place in the progress of arterial degeneration is the diminution of the capillary field. This obliteration of the capillaries is likely to be found of the greatest importance not only in the production of the degenerative changes that occur in the heart itself, but by narrowing the communication between the arterial and the venous system it entails more work on the heart in forcing the blood through the constricted area.

If one notes the changes in the skin that occur with advancing years,

how it loses its velvety thickness, becomes shrivelled and attenuated, so that in advanced conditions the scalp may be found denuded of hair and plastered to the underlying bones, the extent of the diminution of the capillary field may, to a certain extent, be appreciated. A still more striking evidence of the diminished capillary field in the old is the absence of free bleeding in a freshly-made wound. In the young the abundant oozing of bright red blood is a source of satisfaction to the surgeon, for it is a testimony to the healthiness of the subject and to the recuperative power, and is in striking contrast to the bleeding from a wound in the aged, where the bleeding is mostly from some cut vein or from the persistent spouting of a degenerated artery, indicating an impoverished blood-supply rendering the healing process less satisfactory.

This diminution of the capillary field, so easily recognized in the external body-wall, also occurs in the heart, and the results are shown in a variety of ways. It leads to malnutrition of the tissues and degeneration of the heart muscle. The character of the degeneration varies according to the structure affected, but in all it leads to impairment of function. The first structures to show evidence of the capillary obliteration are those that have the smallest blood-supply, and it may be partly for this reason that it is early marked in the cornea (arcus senilis), the valves of the heart, and the arterial walls.

In the heart muscle the effect of these changes in the arteries and capillaries is a degeneration, fibrous or fatty. In the production of this myocardial degeneration we get the diminished capillary field complicating the consequences of the degenerated artery—a degeneration at times so extreme that little blood can penetrate the coronary arteries or their branches. If it be borne in mind how dependent the muscular structure of the heart is upon an abundant supply of blood, it will be easy to recognize the fact that such changes must have a profound effect upon the efficiency of the organ.

Accompanying the arterial degeneration, Savill[429] and Russell[427] have shown a great increase in the muscular coat of the smaller arteries. This hypertrophy implies during life abnormal contraction (hypertonus of Russell). This is bound to raise the blood-pressure and embarrass the heart.

The diminished capillary field has also probably a further complicating effect in so far as it introduces an obstruction to the heart's contraction. The narrowing of the outflow necessitates a greater force to send the blood through the tissues ; consequently the ventricle has to contract more strongly to raise the arterial pressure, and thus produces a further embarrassment to the degenerated heart.

§ **222. Symptoms of cardio-sclerosis.**—The symptoms arising from

such changes are extremely varied, and at first sight hopelessly confused, but there is good reason to expect that with a better knowledge of the functions of the different parts of the heart a more satisfactory appreciation of all the symptoms may be obtained, and, in turn, a more accurate understanding of the symptoms during life will guide the pathologist in his post-mortem examination. I have submitted to Professor Keith a large number of hearts affected by the changes associated with arterio-sclerosis from patients ranging from forty-two to seventy-seven years of age, and in all the post-mortem appearance had such a close resemblance that it might have been assumed that during life the symptoms would have been identical. A study of these symptoms showed, however, a wide diversity, some patients suffering from angina pectoris, others with no pain ; some with severe cardiac asthma, others with no respiratory trouble ; some had very irregular hearts, others frequent or infrequent extra-systole, while some had marked pulsus alternans, and in others the heart was perfectly regular till the end. Some patients had extensive dropsy, other patients showed no sign of oedema. Some had aortic or mitral murmurs, others had no murmurs. It will thus be seen that the symptoms of well-authenticated cardio-sclerosis exhibit every phase of cardiac symptoms, and the superficial observer might think that each case presented a different form of heart disease. Instead of this, while the organic or fundamental lesion is the same, the variety of symptoms is due to the different parts or functions particularly affected.

The earliest result of cardio-sclerosis is a diminution of the reserve force of the heart, manifested by a limitation of the field of cardiac response. The patient rarely presents himself before the physician until this exhaustion of the reserve force has produced some distressing symptom, it may be breathlessness, cardiac asthma, angina pectoris, or ‘ bronchitis ’. In every case it will be found to be preceded by a history of an ever-diminishing area of cardiac response. At the beginning the individual will not acknowledge that his powers are being curtailed—indeed the patient may be proud of his virility—but it may be taken as a certain sign that when a middle-aged man boasts of his strength he is trying to hide from others his own consciousness of a limitation of his powers. Continuing to work as hard as he did before these degenerative changes made their appearance, the exhaustion of the reserve force, though slight at first and scarcely perceptible, in the long run reaches a stage when the suffering or discomfort entailed compels the patient to consult his physician. When this occurs the changes in heart and blood-vessels are well established. The skin of the hand has already lost its velvety thickness, and the arteries show a varying degree of change, as tortuosity, slight or considerable thickening of the radial ; sometimes one can detect

pieces of peculiar hardness, fine and granular, or patches like small beads, or the artery may be thickened like the characteristic pipe-stem, the surface being slightly nodulated.

Even in the absence of any of these signs in the superficial arteries, it must not be inferred that the degenerative process is absent in the visceral arteries. Arterial degeneration is often very irregularly distributed, affecting different regions in different patients. It is for this reason that in this affection the symptoms of its progress may be more marked, now in the cerebral arteries, giving rise to cerebral apoplexy, now in the arteries of the leg, giving rise to gangrene, now in the arteries of the heart, giving rise to the symptoms here described.

The blood-pressure measurements show, in many cases, a great rise. In the earlier stages, when the patients are first seen, there is seldom much enlargement of the heart unless there has been long-standing Bright's disease. Usually the heart's dullness does not extend beyond the nipple line. The sounds of the heart may be clear and well struck, often with some accentuation of the second sound. In some an aortic murmur may be present, most frequently systolic in time, though occasionally there may also be a diastolic murmur, usually of very short duration. The heart's action, though frequently perfectly regular until the end, may show irregularities, the most common being of the nature of extra-systole. In advanced cases we may find good examples of the pulsus alternans. The heart may be continuously irregular (nodal rhythm), and sometimes of great rapidity. Not infrequently this continuous irregularity, with or without excessive rapidity, comes on in intermittent attacks lasting for a few minutes, a few hours, or a few days (paroxysmal tachycardia). In rare cases the degenerative process may affect the a.-v. bundle and give rise to heart-block.

The subjective phenomena vary. In the early stages there may be no symptom beyond a limitation of the field of cardiac response, shown by breathlessness on moderate exertion. In more advanced cases there may be a slight tightness across the chest on exertion or on going into the cold air, as on going from a warm room into a cold bedroom, or going into the open air on a winter's morning. This sensation is usually ignored until it is accompanied by pain, sometimes of such severity as to be recognized as an attack of angina pectoris. In rare cases the pain may never arise, but the gripping sensation felt in the chest may be so severe that the patient feels his chest fixed, and has to stop and draw several deep breaths to relieve the spasm.

In many cases it is only breathlessness on exertion that arrests the patient, the breathing being short and hurried on such exertion as he used to undertake in comfort. In extreme cases the mere turning over in bed induces

the hurried respiration. The breathlessness may seize him in the night—in attacks of cardiac asthma, or Cheyne-Stokes respiration may appear.

The symptoms described so far arise from the heart while the tonicity is still good. In a great many of these cases a stage is reached when the heart dilates. In addition to the increased size of the heart, a number of symptoms disappear, while others come into prominence ; the arterial pressure falls, the attacks of angina pectoris, cardiac asthma, and Cheyne-Stokes respiration usually disappear, while a mitral systolic murmur may be heard ; dropsy sets in, and oedema of the lungs, sometimes with the expectoration of blood, or blood-stained sputa—in short, all the symptoms already described under failure of tonicity (Chapter XXIII).

If the cause of the symptoms in cardio-sclerosis is appreciated, it helps one to understand the reason of the great variety of phenomena present in this affection. The variations in symptoms are in all likelihood due to the parts invaded by the degenerative process. As the presence or absence of aortic murmurs depends on whether the disease affects the aortic valves, so the presence or absence of the various irregularities (with the exception of the pulsus alternans) depends on the invasion of the primitive cardiac tissue. The extent of the invasion determines whether the irregularity is limited to an occasional extra-systole, or whether it terminates in the nodal rhythm or in heart-block. In like manner the degree of exhaustion of the function of contractility determines the nature of the subjective phenomena, the breathlessness, anginal symptoms, and the cardiac asthma. On the other hand, with exhaustion of the tonicity we get the transformation in the character of the symptoms resulting in the dilatation of the heart, dropsy, oedema of the lungs, and so forth.

§ 223. **Prognosis.**—The prognosis depends to a great extent on the nature of the symptoms, and the manner in which the heart responds to treatment. If, for instance, a patient has an irregular pulse due to extra-systole, while in other respects the response of the heart to effort is such as would be expected under normal conditions at his time of life, then the prognosis, in the absence of other evidences of disease, is very favourable. When graver symptoms are present, as the tightness across the chest, slight or severe attacks of pain, then, if the previous history of the patient points to worry, sleeplessness, and overwork, a prognosis should be deferred to see how he responds to treatment. If these signs speedily disappear under treatment, the prognosis is favourable ; on the other hand, the prognosis becomes the more unfavourable the more the symptoms refuse to yield. But even here comparative freedom from suffering may be enjoyed by a patient who pursues a life that exposes him to little effort, and many patients may live a useful

though crippled life for years. Where there are attacks of cardiac asthma occurring in the night, or attacks of Cheyne-Stokes respiration, or when the pulsus alternans is present, the condition may be considered far advanced, and though the patient may live for months or a few years, it is with very limited powers, and he is liable to a serious breakdown at any time. When the pulse is continually irregular, the prognosis depends on how well the heart maintains the circulation. If dropsy supervenes and steadily increases, it is not very susceptible to treatment in contrast to the parallel condition due to rheumatic sclerosis. But apart from this, many patients may lead a fairly active existence with the nodal rhythm for many years, though exposed to frequent attacks of ' bronchitis '.

It must be borne in mind that the sudden inception of the nodal rhythm is not infrequently the direct cause of death in elderly cardio-sclerotics.

The rather rapid dilatation of the heart, with accompanying dropsy, is usually a sign of approaching dissolution.

I doubt if the blood-pressure measurements prove of much use as a guide in prognosis. I have watched for a number of years individuals glide past seventy years of age with a blood-pressure from 180 to 200 mm. Hg., and I could not see that their condition was materially worse in consequence.

§ 224. **Treatment.**—In treating cardio-sclerosis it should always be borne in mind that the condition is progressive, and we cannot stay it, because the changes are those which are inseparable from advancing years. It usually proceeds very slowly, so that a man may show signs of arterial degeneration and irregularity of the heart from the age of fifty to sixty years, but may live in fair health for twenty years afterwards, ending his days without any marked failure of the heart. The early stages are generally recognized in the examination of the patient for some other condition, when the distinctive signs may be found in the tortuous arteries, raised blood-pressure, and the occasional occurrence of an extra-systole. Medical men often attempt to combat these signs by some treatment more or less energetic, and as many people are frightened by the evidences of advancing years they readily comply with the proposals that are supposed to put back the hands of time ; hence the great variety of drugs, methods, and modes of life we find current.

It is rare that one has occasion to treat the milder symptoms in the working-man—not that they are not frequently present, but because he has not the time to consider his complaints, and he seems in no way to suffer from the neglect. It is the well-to-do who are most concerned about some trifling symptom incidental to these changes, and when their attention is called to such a symptom as an extra-systole, either by their own sensations

or by their medical attendant, they believe that some calamity is impending, and readily submit to any suggestion that promises to stave off the evil day.

When the patient is aware of the irregular action of his heart, and when we find by examination that there are no changes beyond what would be expected at his time of life, he should be strongly reassured that the irregularity is a trivial symptom and of no vital importance. When the symptoms are unpleasant and are aggravated by his mode of life—for example, by too close application to a sedentary occupation—certain rational suggestions as to the manner of living are obligatory.

In cases presenting these milder signs no further treatment is necessary, beyond insisting that the patient should lead a well-regulated life, avoiding over-feeding, over-drinking, and taking as much exercise in the open air as can be reasonably obtained. Some of the symptoms, as extra-systole, come on for periods and disappear for longer and shorter intervals. In these cases I have frequently seen the patient get much benefit from a holiday with healthy open-air exercise. Several of my patients, for instance, were conscious of the occurrence of extra-systoles, and when they felt them they indulged in a game of golf two or three afternoons a week, or took a short golfing holiday, and invariably experienced relief. In like manner, judicious hill-climbing and walking are of benefit. In some cases the bodily exertion may in the first instance increase the frequency of these extra-systoles, but the exercise should not be given up on that account; rather should it be continued with moderation till a recovery of the reserve force takes place by training, when the irregularities will become less frequent or disappear.

The amount of recovery depends on the stage which the degeneration of the heart muscle has reached. We know of no method which can restore a better blood-supply by removing the arterial degeneration, and without this it is impossible to arrive at any process that would restore the degenerated muscle of the heart; so that when a considerable degree of recovery has taken place, it is foolish to imagine that the favourable result has come to pass because the treatment has restored the degenerated muscle-fibres. All we can say in such cases is that the treatment has increased the reserve force of the muscle-fibres. Recovery means the retention of a certain amount of active muscle-fibre, and the greater the recovery, the less the degeneration, and the less serious the prospect for the patient.

Another most important factor in treatment in all these cases is sleep. Many suffer from troubled and broken sleep, and when they begin to suffer from attacks of heart failure the occurrence of sleepless nights almost invariably precipitates the exhaustion. Attacks of angina pectoris may be directly induced by this want of refreshing sleep, and may be stopped by measures

taken to induce sleep. The means best adapted to this object varies with different individuals. In some it may be found that their former habits in the matter of their food are no longer suitable for their condition ; it is sometimes enough for them to take some light nourishment, as milk or biscuit, on going to bed or during the night, to induce a restful sleep. Most require some form of hypnotic, and the bromides—20 grains thrice daily, for instance—may induce a degree of drowsiness that is very beneficial. Again, the safer hypnotics, as veronal or sulphonal, prove very useful in the milder cases. When, however, the nights are disturbed by attacks of distressful breathing, oxygen is of great service in some cases, and in others opiates or chloral must be resorted to. On the whole, I find chloral the more useful drug. But the cases are so variable that sometimes one drug is more efficacious than the other, so that it may be necessary to try each of them, or a combination of both. As regards contra-indications, I do not prescribe opiates when there is exudation in the bronchial tubes with duskiness of the face, as I have seen serious results follow, probably from the secretion not being got rid of and thus inducing a certain amount of suffocation, which further impairs the enfeebled heart.

Iodide of potassium is now generally recognized as of service in the relief of many of the milder symptoms associated with arterial degeneration. In many people the symptoms are not constant, but are manifested occasionally in attacks of dizziness, dull headaches, inability to walk as far as usual on account of breathlessness, slight attacks of angina pectoris, and even more violent attacks ; these all seem to benefit by the use of iodide of potassium. In the attacks of ' bronchitis ' so common in the winter and spring months in certain classes I have found distinct benefits from the use of the iodide. I am not at all sure that the good results attributed to the iodide may not have been due to the accompanying change in the food and mode of life. The action of iodide of potassium is not understood.

When dilatation of the heart sets in, with dropsy and scanty urine, the treatment should follow the lines laid down in Chapter XXXIII. I may remark here that drugs of the digitalis group are frequently of little avail in these cases. They may increase the flow of urine, but the heart does not readily respond to them.

§ **225. Aneurysm.**—It is not my purpose to enter into the discussion of aneurysm, as it comes within the province of arterial disease, with which I do not propose to deal. As, however, aneurysm is a frequent complication of cardio-sclerosis and adds to the embarrassment of the heart in its work, I wish to point out that many of the symptoms present in aneurysm really arise from the heart, which has become affected by the processes that have

led to the production of the aneurysm. The aneurysm itself is such a patent abnormality, while the heart affection (cardio-sclerosis) may give rise to no distinctive sign, that the cardiac symptoms are usually attributed to the aneurysm. Thus the pain in aneurysm is very often cardiac in origin, especially when it occupies the region illustrated in Fig. 12. There are, however, other pains in aneurysm whose origin it is difficult to indicate, such as the persistent boring pain felt at the back over the left scapula, or on the top of the shoulder. Whether they are the direct result of the pressure on sensitive structures, or are of the nature of referred pain, I cannot decide.

The majority of cases of aneurysm do not die from rupture of the aneurysm, but from exhaustion of the heart. This is important to remember, for the treatment is often directed to the aneurysm, whereas it is the heart that needs to be looked after.

CHAPTER XXVIII

Adhesive Mediastino-Pericarditis

§ **226. Etiology.**—The adherent pericardium secondary to rheumatic pericarditis rarely gives rise to any sign. In these cases the pericardium is not adherent to structures outside the heart. On the other hand, certain obscure inflammatory affections, probably of a tubercular nature, such as occur in 'polyserositis', give rise to very marked phenomena. There is an extension of an inflammatory process which affects all the structures in the mediastinum, welding together the heart and pericardium, and firmly binding these to all the surrounding structures. The heart becomes anchored to the spinal column behind and to the chest-wall in front. As the spinal column is unyielding, the contracting heart pulls on the ribs in front, and as they yield to a greater or less extent we find the ribs drawn in during systole and springing back during diastole. This embarrassment of the heart leads to great enlargement in its size, and some of the biggest hearts met with are due to this disease.

§ **227. Symptoms.**—The patients are always very short of breath, and usually have to be propped up when in bed. As a rule, little or no pain is complained of, but in one case I found that attacks of angina pectoris of the most severe form were easily provoked. A slight effort would bring on an attack, especially if the patient laughed. Attacks could sometimes be induced by pinching the skin under the left nipple, or by applying the stethoscope.

The adhesion of the heart to the lungs, blood-vessels, and other surrounding structures produces a great variety of symptoms, the cause of many of which is obscure. The chief symptoms are great enlargement of the heart—so great sometimes as to cause a marked difference between the two sides of the chest—and retraction of the structures surrounding the heart during ventricular systole. The systolic retraction alone is not distinctive, as I have shown that it occurs when the anterior surface of the heart is made

up of the right ventricle (Fig. 33). During the ventricular systole there is often an indrawing of the lower intercostal space on the left side behind (Broadbent's sign, Fig. 150). Tallant and Cooper have shown that this may arise in enlargement of the heart (with compression of the lungs) without pericardial adhesion. In such cases, however, the interspaces affected vary with respiration, and Cooper suggests that when they do not vary

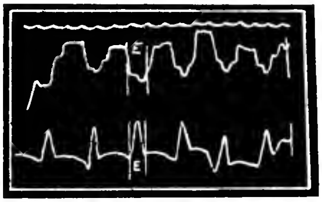

Fig. 150. The upper tracing was taken from the 9th left intercostal space behind from a case of adhesive mediastinitis, and shows 'Broadbent's sign', which is seen to be an indrawing of the intercostal space during ventricular systole (space E).

with respiration the sign is, as Broadbent states, an evidence of pericardial adhesion. When the chest-wall is thin and the heart is not covered by lung, the systolic retraction of the different interspaces can be seen in a peculiar wave-like rhythm.

Though various murmurs and modified sounds are often heard, no distinctive sign can be found on auscultation. The veins of the neck may sometimes be seen to swell up during inspiration. A very curious symptom

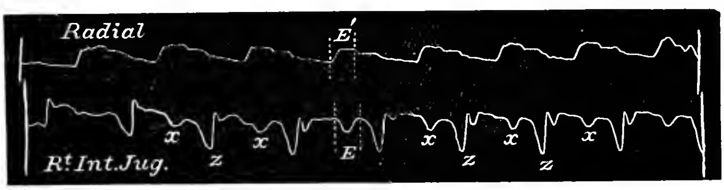

Fig. 151. Simultaneous tracings of the radial and jugular pulses, showing a great depression (z) occurring during the ventricular diastole. (From a case of adhesive mediastinitis.)

is a sudden collapse of these veins at the beginning of diastole, which Friedreich has explained as due to the springing back of the ribs after the ventricular systole has dragged them down, the cavity of the thorax being thus suddenly enlarged and expediting the flow from the overfilled veins. This is well seen in the jugular tracing, Fig. 151, where the fall z is due to the diastolic expansion of the chest.

The radial pulse may show a diminution in size during inspiration—the pulsus paradoxus. It is now recognized that a respiratory variation of the

pulse may occur in a great variety of conditions, but I think that in adhesive mediastinitis presents certain distinctive features. Curiously enough, no tracings have been given, so far as I know, showing the pulsus paradoxus in adhesive mediastinitis along with the respiratory curve, except in two instances taken by Nicholson from Gibson's [398] cases. The study of these tracings, compared with some I have taken, leads me to think that the variations point to very different causes. As, however, my observations are few in number, I do not enter into the subject, but call attention to a field that needs exploring.

There is usually associated with these signs enlargement and sometimes pulsation of the liver, which in Wenckebach's [437] case was of the auricular type. The spleen also may be greatly enlarged, and there may be considerable dropsy.

§ **228. Prognosis.**—The future of these cases is bad, though they sometimes show periods during which they make remarkable progress towards recovery from serious symptoms. But these are only temporary, and they gradually drift to a fatal issue.

Treatment.—The treatment of these cases has so far been unsatisfactory, and one can only advise the principles usual in extreme heart failure. Attempts have been made, following the suggestion of Brauer, to free the heart by resecting the ribs. This has been done in several cases, and Wenckebach [437] describes marked improvement in a case in which the operation was performed.

CHAPTER XXIX

Congenital Affections of the Heart

§ **229. Etiology.**—Congenital heart affections are due to the persistence of certain foetal forms of the circulation, such as persistent patency of the foramen ovale or ductus arteriosus, or to some interference with development leading to deformation of the valves or narrowing and obliteration of the large arterial trunks. They may also arise in consequence of foetal endocarditis. The conditions are incompatible with life in many cases.

It is only in exceptional instances that the symptoms permit of a recognition of the nature of the cardiac defect.

§ **230. Symptoms.**—The most characteristic symptom is cyanosis, which is present in a great number of patients. Clubbing of the fingers is a common accompaniment of the cyanosis. The size of the heart is often greatly increased. This may be due to hypertrophy of the left ventricle when there is an obstruction to the outflow of the blood through the aorta, or to dilatation of the right heart when there is interference with the pulmonary circulation or a patent foramen ovale. Murmurs are usually present, almost invariably systolic in time, but it is difficult to detect their origin except in the case of the patent ductus. Here the communication between the aorta and pulmonary artery persists, and as the pressure is much higher in the aorta a continuous stream passes during the whole cardiac cycle from the aorta to the pulmonary artery, and, as Gibson[397] has pointed out, this leads to a murmur which, beginning with great intensity at ventricular systole, extends over ventricular systole into diastole, fading away towards the end of diastole. This murmur is loudest over the second and third left interspaces, and here also a well-marked thrill synchronous with the murmur can be felt.

§ 231. **Prognosis.**—If there be no cyanosis, little or no enlargement of the heart, and the development of the child good, with a fair field of cardiac response, then the prognosis is good ; otherwise the outlook is bad, though the child may lead a crippled existence for many years.

§ 232. **Treatment.**—If the heart maintains the circulation well, no treatment is required. In more serious cases, beyond attending to the child's comfort and nourishment, special treatment for the heart is of little benefit, digitalis being rarely of value unless there is dropsy.

CHAPTER XXX

Heart Disease and Pregnancy

§ 233. **Importance of the subject.**—It is almost certain to be the experience of every practitioner who has a midwifery practice to be met by the question of pregnancy in women with some affection of the heart, and this question is of supreme importance, for life and death depend upon the views held by the doctor. If the gravity of the question is not sufficiently appreciated, the consequences in certain cases may be ruined health or even the death of the woman, while a too serious view of other cases may unjustly entail upon the woman an uncalled-for restraint. Hitherto the guide in the diagnosis has been in the main the presence of a valvular murmur, and some doctors, having seen patients with valvular lesions pass scatheless through repeated pregnancies, treat the subject too lightly, while others, who have witnessed the disastrous results of pregnancy, are oppressed by the gravity of the condition.

The treatise on *The Bearings of Chronic Diseases of the Heart upon Pregnancy, Parturition, and Childbed*, by Angus Macdonald, in 1878, is still the best work on the subject, and long experience and observation lead me to agree with him in most points. The researches on this subject have too often been directed to the somewhat barren and academic question as to whether the heart hypertrophies during pregnancy, and the bearing of this on prognosis. If, however, it be borne in mind that heart failure is essentially a question of the integrity of the heart muscle, a better standpoint will be obtained for judging these cases. The valve lesion in the great majority is at the most only an embarrassment to the heart in its work, and one which it may easily overcome. The presence of the lesion is important, however, in another respect, namely, in that it calls attention to the heart and serves to remind us that the disease that injured the valve may at the same time have injured the muscle: our object in examining these cases is to find out the extent of the lesion in the muscle, and how far the valve lesion embarrasses the muscle.

§ 234. **Standards for guidance.**—The essential question in cases of valve disease is the condition of the muscle, and this is determined by the manner in which the heart responds to effort. Additional help will be obtained by noting the size of the heart, its rate and rhythm, and the tendency to oedema of the legs and lungs. Given a fair field of cardiac response with little or no enlargement of the heart, then pregnancy need have no terrors. If the field of cardiac response is distinctly limited, particularly if palpitation is readily induced by exertion, with no oedema, then the opinion should be suspended till the result of treatment is ascertained. If the condition does not improve, then the outlook is not hopeful, and pregnancy should be forbidden.

With the appearance of dilatation and its accompanying symptoms—oedema of the legs and lungs—pregnancy should be forbidden. In doubtful cases, with some dilatation and no swelling of the legs, I have been accustomed to be guided by the tendency to oedema of the lungs. This is readily ascertained by examining the patient after a night's rest, before she gets up. If she be instructed to lie on one side as much as possible, and not to sit up until she is examined, numerous fine crepitations will be found on the first deep inspiration, if there be any tendency to oedema. The auscultation of the lung base should be the first procedure adopted in the examination. If the crepitations are dispelled with the first few deep inspirations, then it may be taken for granted that there is only a slight tendency to oedema, and the case requires care and watchfulness before finally deciding. If the crepitations persist, then the tendency to oedema is so great that pregnancy should be forbidden (see also § 190).

It must be kept in mind that patients with valvular lesion may suffer from most severe heart failure (dilatation, and extensive dropsy), and make such good recoveries that repeated pregnancies may be undertaken with impunity. Here, however, the degree of recovery gives the standard for the judgment, and I mention this lest the knowledge of a bygone heart failure be considered a bar to marriage and pregnancy.

One of the most difficult problems I have had to face has been where a pregnant woman has shown evidence of heart failure, and the question has arisen whether interference is necessary. For one thing, a natural labour entails less strain upon the heart than one artificially induced. In the former case the preliminary preparation and softening of the tissue render the expulsion of the child much easier, even though the head is larger. But, on the other hand, the dropsy may increase to such an extreme degree that no aid can be rendered.

One of my most painful experiences was in the case of a woman of

thirty-five years of age with mitral stenosis, who became pregnant with her first child; when labour set in the dropsy had become so extensive, and the breathlessness so great, that she could not lie down, but had to sit up in bed. It was impossible to make a vaginal examination, as she could not turn to one side without great suffering. She could not take chloroform, as the inhalation intensified the dyspnoea, and we were helpless, and had to see her die after thirty-six hours of suffering, relieved slightly by opium. In other cases where the dropsy and breathlessness were threatening to become extreme, I have successfully induced premature labour. Fortunately, many women with advanced heart failure abort. In others abundant bleeding from a varicose vein gives great relief, and suggests venesection when there is much evidence of over-filling of the venous system.

The nodal rhythm should be a bar to pregnancy in all cases.

Although the valvular lesion by itself is not a contra-indication, yet the particular valvular lesion influences the decision when it is combined with indications of muscle failure. Thus aortic lesions are very serious unless there be a good intact heart muscle. Mitral stenosis, when there is only a presystolic murmur and good effective muscle, is no bar; but if there is also a diastolic murmur, then, unless the muscle is very good, pregnancy should not be permitted, as the diastolic murmur points to a progressive narrowing of the orifice.

§ 235. **Management of the labour.**—In regard to the management of labour in these patients with heart affections, if there is the slightest sign of heart failure the patient should be instructed not to bear down, and chloroform should be administered very early, the anaesthesia being gradually deepened. As soon as it is feasible, forceps should be applied, and gentle, firm traction intermittently maintained until delivery is effected —even if the forceps have to be on for a considerable time.

I have never seen the slightest risk from chloroform in these cases.

After the delivery is over the patient is not out of danger, for symptoms of severe heart failure may supervene any time within the next three weeks. In view of this, care should be taken to ease the heart's work, by rendering the patient comfortable and inducing refreshing sleep. In severe cases nursing the child is out of the question, nor should the child be allowed to disturb the mother. The judicious administration of digitalis is of service, following the lines laid down in Chapter XXXIV.

CHAPTER XXXI

Chloroform in Heart Affections

§ 236. **Conditions contra-indicating its use.**—For a number of years I made numerous careful observations on patients to see if I could detect any special action of chloroform on the heart. I got various alterations in the pulse-rate, and in the degree of venous engorgement, but these were in the main due to the patient being excited or struggling, or holding his breath, so that I could detect no sign of any direct influence of the chloroform on the heart. It must be borne in mind that a great many of the physiological experiments have been carried out with very large doses, and with the animal's heart in a very unnatural condition, so that the results cannot be compared with those of the small doses given to a human being.

The question whether a patient can take chloroform has often to be met, and the indications against its administration have never been clearly given. The stethoscopic examination of the patient just before administration is useless; the decision must be made independently of any results obtained by it, for no form of murmur, irregularity, or rate is a bar to the administration of chloroform. I am not aware of a single form of heart ailment discoverable by auscultation that would render the administration of chloroform dangerous. The dangers do not lie in the action of chloroform upon the heart alone, but on other considerations. Thus, there is often trouble and even danger in giving chloroform to emphysematous people with great liability to bronchitis, and to elderly people with a tendency to bronchitis and with wheezing râles accompanying respiration, and also in cases of embarrassment of the lungs from oedema, abundant pleural effusion, or tumours pressing on the windpipe—in fact, all cases with imperfect oxygenation of the blood.

Elderly people with a suspicion of cardio-sclerosis are also bad subjects, not because of the chloroform, but because their hearts are unable to withstand even slight strains. Thus one typical cardio-sclerotic who was to be operated upon for piles, and who had suffered from intense irritation about the anus, and was most anxious to be relieved, had shown signs

of slight attacks of angina pectoris. He was greatly excited on entering the operating room, and when laid down preparatory to receiving the chloroform his pulse was very rapid. The towel had just been placed over his face when his pulse stopped. The towel was at once removed, and he continued to respire with considerable force for a few minutes and then stopped, and he was dead. Manifestly it was not the chloroform that killed, but the strain on the enfeebled heart.

The Status Lymphaticus.—Of late years, a number of deaths have occurred in a condition described as the status lymphaticus. I have no experience of this condition myself, but it is described as a state in which the sufferer may die from any trivial cause, and which is marked by enlarged thymus and spleen, hypertrophied lymphatic glands in various regions, with swelling of the tonsils, and of the solitary follicles and Peyer's patches in the intestine, and flaccid cardiac muscle. The condition may be recognized during life from the pale thin skin and pasty complexion and fairly abundant subcutaneous fat, along with frequent signs of rickets or scrofula, enlargement of the tonsils and of the superficial glands, especially in the axilla and neck, adenoid growths, a palpable spleen, and often enlargement of the thyroid (McCardie).

§ 237. **Estimation of the fitness of the patient.**—It will be seen that the examination of the patient should be made prior to his reaching the table, and if there is a doubt, even a day or two before the operation. Apart from the status lymphaticus, the best test is the patient's field of cardiac response. But even when the field is greatly limited from some exhausting disease, with or without valvular affection, the patient may take chloroform with safety. In these circumstances, the operation is sometimes delayed for days or weeks in order that the heart may be strengthened by drugs like strychnine and digitalis. Now this is very bad practice, for it may be taken for granted that no patient's heart will gain strength while an operation is hanging over his head, more particularly if he or she knows that the operation is delayed for the heart to be strengthened. I can conceive of nothing better calculated to weaken the heart than such a procedure, for the anxiety is bound to react upon the heart and make it more irritable. When a patient with a weak heart is to be operated on, the sooner it is done the better.

Under the influence of excitement and fear, the patient's heart may appear worse than it is, and one should always keep that in mind when the heart is behaving strangely before an operation, as the following illustration shows. I proposed to remove a large ovarian cyst from a very intelligent patient ; all arrangements were made, and when I arrived in the morning

to do the operation I was informed that the patient had gone out of her mind, and that her heart was very bad. When I saw her she was muttering incessantly, and took no notice of any question I put to her. Her pulse was extremely small and very rapid, 160 beats per minute. After reflecting over the case I concluded that the condition was in all likelihood due to apprehensive fear, and that chloroform would be likely to soothe the brain and relieve the heart, so that to delay the operation would but add to the terror of the patient when she recovered. I therefore asked the anaesthetist to proceed, but he refused, and I started the chloroform myself. She went under very quickly, and the pulse speedily fell to seventy beats per minute, at which rate it remained during the operation, and when she came out of the chloroform she was perfectly rational, and made a good recovery.

I have administered chloroform repeatedly in cases of extreme heart failure with valvular lesions—for example, during labour, and in one case where the patient appeared to be dying from a severe attack of pneumonia—and I have never seen the slightest bad effect from the chloroform. Cases with extreme irregularity of the heart can take chloroform without danger. I watched for a whole hour a case of complete heart-block under full anaesthesia.

The cause of death under chloroform anaesthesia—apart from overdose and imperfect aeration of the blood—has so far escaped recognition. It is probable that the secret lies in the fact that certain changes, as yet unknown, render some hearts abnormally susceptible to the action of chloroform. One can infer this from the effect of digitalis, which is shown in Chapter XXXIV to have a varied action depending on the nature of the heart lesion. At all events, no satisfactory conclusion will ever be reached until graphic records of the movements of the circulation and respiration are made during the whole time of anaesthesia. As death occurs quite unexpectedly, it would be necessary to make observations consecutively on many thousands of cases.

CHAPTER XXXII

Prognosis

§ **238. Responsibility of the medical profession.**—In addition to recognizing the meaning of any abnormal sign or symptom, we should endeavour to acquire a knowledge of what bearing it has upon the future history of the patient. This knowledge can only be obtained by watching how patients exhibiting the abnormality meet the storm and stress of life. This has been a special object of my work on the heart for over a quarter of a century, and in the following observations I am culling from my own personal experience, and in each deduction I give I have in my mind a number of cases from which it has been drawn.

I am rather afraid that our profession as a body does not recognize sufficiently its responsibility in regard to prognosis. When an individual submits himself for an opinion, he does so with such implicit confidence that the verdict given may alter the whole tenor of his life. He may be, for instance, seeking to enter some profession, when a preliminary medical examination reveals what the medical man takes to be an abnormality. An imperfect knowledge of its nature may, and unfortunately often does, lead to its being regarded as presaging possibly grave consequences, and the candidate is rejected. He is thus shut off from the prospect of his chosen calling, and, knowing the reason of his rejection, passes through life uneasily conscious that some disaster is always impending, while all the time the supposed abnormality may be a sign of little or no consequence.

If we look at an insurance paper we realize the hardships to which an applicant is exposed. Is the pulse regular? Are the sounds pure? If either question is answered in the negative, the applicant is either rejected or is penalized for life by having to pay a higher premium, and, in addition, is burdened with the painful consciousness of infirmity.

I dwell on this matter with some insistence, because I have known of so many instances in which gross injustice has been done to individuals, not only in the pecuniary aspect, but in having imposed upon them great expense, unnecessary treatment, and mental disquiet, because the meaning

and prognostic significance of some simple symptom, as a murmur or an extra-systole, have not hitherto been recognized. I sometimes wonder whether the use of auscultation has not been the means of doing more harm than good. That it is not an unalloyed blessing is too painfully evident, for not only have totally incorrect conclusions been drawn as to the bearing of murmurs on the future of the patient, but so much time has been spent in investigating the physics of their production that more important matters have been lost sight of. It is so easy to recognize a murmur, that other less obvious but more significant signs have too often been neglected.

A serious responsibility is thrown upon every practitioner at times in advising upon other questions. Should a man give up his business ? is a question on which advice is constantly sought, and whether the individual be a statesman or a labourer the greatest care is necessary in formulating the answer. Should a woman with some heart affection marry, or, if she is pregnant, should the pregnancy be allowed to proceed ? are problems that every general practitioner at one time or another will have to meet ; and if he seeks for guidance in the textbooks he finds merely general views which he cannot apply to the individual case. This fact alone should arrest the attention of the profession, and make it conscious how inefficient the teaching of heart affections has been.

§ 239. **Basis for prognosis.**—A rational prognosis must be based on a clear idea of the manner in which any given symptom is produced. Knowledge never dispelled the terrors of darkness with more effect than in showing the true meaning of the symptoms in affections of the heart. So impressed are the public and profession with the suddenness with which death may take place, that an unnecessary fear lays hold of them when the heart shows any sign out of the common, lest this should be the thing that slays. It is because of this that I have entered with such fullness into the explanation of so many symptoms. I confess there are still many which I do not understand, but I have endeavoured to find out their value by watching individuals who exhibited them, and to find a basis on which their value can be estimated.

In estimating the value of any abnormal sign, or in determining the condition of a heart, the most reliable guide is the manner in which the heart responds to exertion. This, again, is but an attempt to estimate the amount of reserve force. If the individual can with comfort make such exertions as we would expect at his time of life, then the abnormality may with certainty be assumed to be of little real significance.

If there be a complete breakdown, the decision should not be made until time has shown to what extent recovery takes place. The amount of

recovery enables us to judge the condition of the muscle of the heart, for it is on its capability to renew its reserve force that the future of the patient depends. To illustrate this I cite the history of a man whose case is also described in the Appendix for other reasons (Case 17, Appendix IV).

In 1883, at the age of thirty-two, the patient had a severe attack of rheumatic fever, and was left with a mitral lesion. In 1897 he lay for weeks unconscious and swollen with dropsy. From this he recovered with a well-marked presystolic and diastolic mitral murmur and a slightly enlarged heart. In 1904 his heart became continuously irregular, and has remained so ever since. It now extends two inches beyond the nipple line, and he has a systolic and long diastolic mitral murmur. Notwithstanding all this, for the past four years he has followed his trade as a mechanical engineer, doing the very hardest work of this laborious trade, and from 1904 to 1906 he worked overtime almost daily. Beyond being somewhat short-winded going uphill, especially in cold weather, he feels as fit for work as ever he did.

Even cases that never show so complete restoration of function as this, and in which attacks of extreme failure are frequent, may go on for many years and lead sometimes fairly useful lives, though in time the progressive changes become so great, or the muscle so exhausted, that the possibility of even temporary recovery is precluded.

In individuals in whom there is a distinct limitation of the field of cardiac response, a close scrutiny should be made into the cause. It should be borne in mind that if a heart is not properly exercised its field of response becomes more and more restricted. Thus a man who for a long time leads a sedentary life is often startled by the fact that he is rendered extremely breathless by undertaking some exertion that he was wont to make with ease a few years previously. But with moderate training there is soon restored sufficient reserve force to enable him to perform his task without distress. Therefore in all cases, even when there is an abnormal symptom—as a murmur or an irregularity—this question of the nature of the exhaustion should be borne in mind. It must not be forgotten also that the supposed abnormality may have nothing to do with the symptoms of exhaustion. This is particularly the case in the young, in whom syncopal attacks are not infrequent. I have repeatedly seen grave alarm aroused because a boy or girl has fainted, and has had an irregular pulse when quiet in bed. This irregularity has been of the youthful type (sinus irregularity), and if it had any connexion with the syncopal attacks it was merely incidental, and in no sense added any gravity to a trivial affection.

While the lines on which prognosis is based can be fairly well recognized

in regard to the more common affections of the heart, we often meet with patients who show symptoms whose nature is too obscure for us to identify. A prognosis in these cases is often required and difficult to give. The plan I have adopted is to exclude the possibility of degenerated muscle by an analysis of the condition of the separate functions of the heart muscle, and consider how far the complaints may be nervous in origin; having satisfied myself that the muscle is sound, I give a favourable prognosis, at the same time indicating the obscurity of the case. I do this because, as a matter of experience, I have found that these exceptional cases, particularly in young adults, always tend to recovery to a greater or less degree. This unfortunately is not the usual plan, for some signs are too often taken to be more serious the more obscure they are. In many cases the physician must be prepared to back up his opinion by taking a grave amount of responsibility. For instance, I have on several occasions seen patients kept in bed and put through elaborate forms of treatment after some such affection as influenza. The patients have complained of obscure signs, to them alarming, and a certain amount of abnormality has been present, as frequent pulse or extra-systoles. Having satisfied myself that there was no serious mischief, I have had no hesitation in making the patients get up and resume their ordinary life, even when the medical attendant has shrunk from the responsibility. I have never yet had cause to regret such a procedure, and it is better to run a little risk in a rare case than to have a patient drifting on to invalidism because of our ignorance and fear of responsibility.

CHAPTER XXXIII

Treatment

§ **240. The essential principle in treatment.**—In order to treat failure of the heart intelligently it is necessary to find out some principle which will serve as a guide. So far as the heart is concerned we get a safer guide than is possible for any other viscus, as the nature of its failure can be more easily ascertained. As in all forms of heart affections exhaustion of the reserve force is the essential factor in the failure, so the restoration of this reserve force is the aim and object of treatment. But in the first place an accurate diagnosis should be made, and if we detect some change which alarms the patient, but is really of little significance, as certain murmurs or irregularities, then the reassurance of the patient as to the innocent nature of his trouble brings relief; or if we detect some permanent change it is useless to waste time and energy in the haphazard prescription of supposed remedies. It is futile to imagine that we can restore a cicatrized valve, a sclerotic myocardium, or a calcareous artery to their pristine condition. We must recognize facts, and, with the knowledge of the presence of such irremediable conditions, endeavour to make the best of the heart muscle that is left.

Whatever may be the nature of the organic lesion, the immediate heart failure in any given case is due to the heart having to undertake work which makes a call upon the reserve force, while the period of rest is not sufficiently long to permit it to recuperate. Step by step the exhaustion proceeds till some striking evidence of this heart failure finally compels the patient's attention. If this conception of the cause of the heart failure be grasped, we are at once placed in a position to undertake a rational treat-

ment; the principle of such treatment is so simple that it seems almost unnecessary to dwell upon it, namely, the placing of the heart in a position to regain its reserve force. This principle is so obvious that it is apt to be ignored, but when any successful form of special treatment is investigated, it will be found that its success is due to the unconscious adoption of this principle.

The next step to be taken after recognizing the principle is to find out what functions of the heart muscle are exhausted. This, as a rule, can be ascertained by methodical inquiry into all the symptoms after the manner I have detailed in the text. The adoption of special measures when necessary will follow the result of this examination into the nature of the failure, and these I have indicated under the description of the different symptoms. Here I will dwell more on the general application of principles suitable to the treatment of the great majority of cases of heart failure.

§ **241. Rest.**—On examination we may find evidence of some organic change in the heart, as a valvular murmur, persistent irregularity, or dilatation, and then we recognize that the heart has not only been doing the work incumbent on a healthy heart, but it has been doing so hampered by its own inherent defect. As we cannot modify the latter condition we must seek to ease the load in the other direction, and it is here that the physician has to exercise a very wise discretion. He should enter into the patient's daily life and find out the circumstances that are likely to induce heart exhaustion. To give up the work by which the patient earns his livelihood may be too serious a matter, but the physician may be able to suggest the omission of certain kinds of work that may relieve the strain without interfering too seriously with the patient's employment. Or it may be that some habit, as over-indulgence in alcohol or tobacco, or some dietetic error, can be corrected with benefit. Besides relieving the heart from overwork, it must be placed in a position to do its work as free from irritation as possible. The great influence which the nervous system has on the heart must always be borne in mind. A worried and anxious mind invariably reacts on the heart, rendering it unstable or excitable. This factor is of such importance that when the question of work arises, if it is found that the cessation of work results in mental worry and disquiet, it is far safer to allow the patient to follow his occupation with discretion, and with due regard to the nature of the work. In severe heart failure, it is scarcely necessary to say, rest in bed is imperative.

§ **242. Sleep.**—Whatever the form the heart failure may assume, sleep is essential. It may be taken as an axiom that if the patient does not get

sufficient sleep he will never get well. Inquiries should be made of every patient in regard to his sleep ; if it is not good and cannot be attained by removing all forms of bodily discomfort, then resort must be had to soporifics. If an individual has been accustomed to some alcohol—beer, stout, or whisky—at bedtime, and the want of it results in sleeplessness, then he should be allowed to have it. The milder hypnotics may be tried, as antifebrin, veronal, or sulphonal, but if these fail resort must be had to chloral and opium. In great restlessness from breathlessness, cardiac asthma, Cheyne-Stokes respiration, these drugs must be carefully pushed until the desired effect has been obtained. As to the selection of the drug, in serious cases I have found chloral the safer and more useful when there is little or no dilatation of the heart, when the blood-pressure is high, as in cardio-sclerosis, and when there is a tendency to ' bronchitis ', opium being contra-indicated in the latter class because of its tendency to prevent the free expulsion of the phlegm.

Among more neurotic patients I have found bromide of ammonium of inestimable service, not only in inducing sleep, but in keeping the mind from worrying, and in inducing a lethargy that is beneficial to the heart. Even in severe cases of angina pectoris I have found it of great value. Thus, a lady with aortic valvular disease, between the age of fifty and sixty, became liable to attacks of angina pectoris. At times these were so severe that she was forced to lie in bed. The exhaustion of the heart was so great that the sounds were sometimes scarcely perceptible. After a period of rest the heart regained strength and she was able to go about, collapsing, however, again and again after a few months. On making minute inquiry into the cause of these attacks, I found that she suffered from sleeplessness for a week or two prior to a breakdown. I therefore prescribed bromide of ammonium, 20 grains three times a day, during one of her attacks of heart failure. In a few days she got quiet sleep and speedily recovered. When she began to experience a restless night she resorted to the bromide, and it never failed to give her sleep, and no doubt warded off the attack, for on several occasions she neglected the medicine and after a few sleepless nights the attack of angina pectoris returned.

The good effects of the bromide in nervous cases have been referred to in Chapter VIII. The dose of the various hypnotics varies with each patient. Small doses should be used at first, and the drug pushed carefully and steadily till some effect is produced. Repeated doses every one or two hours should be tried at first.

§ 243. **Bodily comfort.**—In cases of severe heart failure much can be done by removing all sources of discomfort. Detailed instructions regarding

sponging the body and arranging the bed so that the patient lies comfortably with light and warm bedclothes should be given; too great warmth should be avoided. The patient often wishes to assume some position that the attendant may fancy to be harmful, but his inclination should be complied with and everything done to keep him comfortable in the position assumed, as with the shoulders raised, sitting upright, or bending forward with the head supported. He may prefer sitting in a chair with his arms on a table and his head resting on them. In all these positions he is instinctively assuming a position that helps the heart in its work. In all cases, mild or severe, every source of discomfort from other parts of the body should be attended to, such as an irritating skin affection, piles, frequent micturition.

§ 244. **Diet.**—In calculating the results obtained by any mechanical process it is necessary to understand all the factors concerned. The neglect of one factor necessarily vitiates the calculation. Because of the slight advance in biochemistry many attempts have been made to find a scientific dietary. But as the factors concerned in metabolism are as yet imperfectly understood, it is manifestly hopeless to base a dietary on knowledge that does not include all the factors concerned. Notwithstanding the confident assertion of many dietetic authorities, the perfect dietary has yet to be evolved, and in the meantime we must be content to be guided by common sense and experience.

In cases of heart failure a good deal of harm can be done by injudicious feeding. It must be kept in mind that in extreme heart failure and in febrile cases the digestive functions are themselves greatly weakened, and that to pour food into a weakened stomach is not only to add to the discomfort of the patient, but may produce flatulent distension of the stomach and bowels, which, pressing on the diaphragm, embarrasses the heart and respiration. The manifest weakness of the patient is often taken as an indication for more food to restore the strength, and satisfaction is felt so long as fluid is seen to disappear into the patient's interior. It is very curious how prevalent the custom is, when the stomach is weak, to give it more work to do. The food is prepared in such a manner that the assistance of the mouth is dispensed with, and more work is therefore thrown upon the stomach. Bread and milk, a favourite food, is so prepared that no mastication is needed, and the stomach is burdened with the duty of getting rid of the load. The great importance of oral digestion is not sufficiently appreciated. Not only does the process of mastication in several subtle ways stimulate the digestive glands of other organs, but the juices from the mouth that are mixed with the food not only assist digestion but prevent the flatulence which is so often

such a troublesome feature in the weakened digestion of heart failure. I wish to insist upon this all the more as it is becoming more and more the vogue to give patients partially digested foods, or foods supposed to be prepared to make digestion easier, and, relying upon the notion that digestion is being assisted, the physician is apt to ignore the natural and infinitely better methods provided by nature. These short cuts to treatment are invariably in the end bad for patient and physician—leading the latter to a rule-of-thumb practice, neglecting thereby to make of each individual a special study.

In cases of extreme heart failure, with dropsy, the food should be very limited in quantity—as a rule, small quantities of milk given at frequent intervals, in extreme cases not more than one pint per day. The patient should be encouraged to take a small portion of biscuit, or a dainty sandwich with fresh potted meat, chewed very thoroughly. In febrile cases, or when the mouth tends to become dirty, it should be washed or sponged out, and immediately afterwards a small piece of solid food should be given to chew. The quantities should always be small, so that the patient is not nauseated by the spectacle of an untempting quantity of food.

With less severe cases the food should be more varied, but it should never be forced on the patient. The quantity he can chew is often a very good guide, because if he cannot be tempted to chew much it is manifest his digestive functions are at fault, and it is a very bad practice in such cases to pour in beef-tea and other easily eliminated fluids. The guiding principle should be food, tempting, needing mastication, with little fluid, and that chiefly milk, given small in quantity and at fairly frequent intervals—the intervals depending on the quantity he is able to take. The kind of food should be that which the patient likes, so long as it does not disagree with him. The doctor must be on his guard not to prescribe a dietary suitable to himself, but must bear in mind that what disagrees with him may agree with his patient. In selecting a dietary the resources of an intelligent housewife will often be found to be of much service.

Individuals with heart trouble, but able to get about, should lead a life of abstemiousness, avoiding all excesses. The meals should be small in quantity, and of such frequency that faintness is avoided. It often happens that they become faint in the night, or early in the morning, as they have not broken their fast since the evening meal. A dry biscuit and a small cup of milk at bedtime or in the early morning will often prevent the occurrence of disagreeable sensations.

A class of people for whom many dietaries have been evolved are those who with advancing years show some signs of wear and tear. It may be that

in their vigorous manhood they enjoyed and gratified excellent appetites, but as the years begin to tell the pleasures of the table no longer appeal to them. Signs of the heart failing may manifest themselves, and the individual begins to take thought and seeks advice. Such a one readily becomes the victim of a dietetic craze. A course of life that seems to put back the hand of time appeals to him. As one who has watched many of these patients over periods of many years, I have seen no evidence which convinces me that the various abstemious dietaries that I have tried and seen others try arrest the progress of senility. With advance of years the appetite diminishes as a rule, and this is good, as the process of assimilation also becomes enfeebled. While moderation in all things is good, it is difficult to tell what are its limits.

In some of my cardio-sclerotic patients the appetite has been maintained with remarkable keenness. I have seen such patients becoming seriously crippled through failure of the heart, with the nodal rhythm, very high blood-pressure, and swelling in the legs. I have endeavoured to restrain their appetite and to restrict their diet, but have only succeeded in increasing their weakness and making them miserable. With the resumption of their old dietary I have seen them improve, and glide gently past the threescore years and ten and well on to the fourscore years before they passed away with little suffering. To the dyspeptic, asceticism may appear an ennobling creed, but, as a practical physician doing my best for my patients, I think I would rather see my patients passing the declining years in comfort, even though their chief pleasures were those of the table, than having their lives made tedious and uninteresting through depriving them of that which gives them pleasure in the hope of adding a few months to their existence.

I must also add a warning to those who may imagine they can modify changes in the heart and blood-vessels by the elimination of certain constituents of food, as common salt. I have seen patients made neurotic and apprehensive because a physician had warned them of the evils of common salt, so that they were made conscious of their ailment at each meal, and were filled with dread lest their food should contain salt. The domestic comfort of a whole household may be jeopardized by having to cook the food free from salt, and all the members are made to suffer because of this foolish restriction. This does not apply to patients seriously ill, when, for the removal of dropsy, a special invalid dietary, salt-free, may be tried. The same foolish notion exists in regard to lime, the notion being that lime salts can be removed from the tissues by decalcifying agents, or prevented from being deposited by restricting the lime contents of the food. Calcium given by the mouth is absorbed very slowly and only to

a slight extent, and is as rapidly excreted, while decalcifying agents like citric acid have no appreciable effect.

§ 245. **The condition of the bowels.**—The condition of the bowels should be attended to in every case. Constipation and straining at stool may produce great exhaustion. In the less severe cases of heart affections the habit of going regularly to the closet and patiently waiting will sometimes be effectual. When the bowels are more stubborn, aperients are necessary. The various mineral waters are beneficial, but where the expense is a consideration simpler remedies can be usefully substituted, as a small quantity of Epsom salts in a tumbler of hot water every morning, or a teaspoonful of compound liquorice powder in a tumbler of water at bedtime. A pill containing $\frac{1}{4}$ grain each of extract of belladonna, extract of nux vomica, and aloin, taken three times a day, is in many cases extremely useful. In more stubborn cases enemata may have to be resorted to—a pint of soap and water given at night.

Treatment by purgatives is often helpful in cases where there is much congestion of the liver and abdominal stasis, and scanty flow of urine. A free evacuation can be obtained by any of the purgatives, such as the colocynth and hyoscyamus pill, calomel, or blue pills. After the free evacuation of the bowels, they should be kept open by some milder aperient.

In all these cases care must be observed that the patient is not too greatly exhausted by the movement of the bowels, for it is sometimes surprising how prostrate some patients become after the bowels are moved. An enema may prevent straining, but if the exhaustion is too extreme, the bowels had better be left alone.

§ 246. **The mental factor.**—The consciousness of heart trouble has often a depressing effect upon people, whether the trouble be slight or serious. When such people become convinced that the trouble is curable or not serious, their condition at once becomes greatly improved. Cures by faith, whether in drugs, baths, elaborate methods, or religion, act by playing upon the mental condition. I have already said that we should always study the mental condition of the patient, and its bearing upon his complaint, and we should utilize its peculiar features in treatment. But our employment of this element in treatment should not be the outcome of blind unreasoning faith in some rite or ceremony, bath or drug, but in the intelligent perception of the nature of the symptom. The reassurance of the patient of the harmless nature of the complaint goes a great way in curing him. When there is some affection that cripples him, the reassurance that with reasonable care no danger need be feared is extremely helpful. Even in serious cases, when there is reasonable hope of recovery, or a certain

degree of recovery, the encouragement of the patient may and does help forward his improvement.

This mental factor should, on the other hand, make us extremely chary of giving the patient a gloomy prognosis. There is nothing in my experience so surprising as the manner in which the heart can recover from the seemingly most hopeless condition of exhaustion. And we must bear in mind that a gloomy view may in itself nullify the best attempts at treatment. We should aim at getting the patient into a placid, contented, hopeful frame of mind, so that the heart is not disturbed by emotional reflexes.

§ 247. **Drugs.**—The influence of drugs upon the heart is one to which I have given a good deal of attention, and I have carried out a long series of observations with many of those most commonly employed in practice. The subject is one that needs a great deal more elucidation, and I give here the results of my observations so far.

Many drugs have a reputation for the good effect they have upon the heart, but I found very little evidence of their beneficial action. An exception to this, however, was found in the case of the digitalis group, but even here the manner of action and the cases suitable for treatment have never been clearly described, so that a great portion of my observations were devoted to finding out how the drug acts and how it should be used. The results obtained are so important that I give an outline of them in Chapter XXXIV, with a fuller illustration of some particular instances in the Appendix (VI).

The class of drugs, apart from digitalis, that I found having a more or less demonstrable effect upon the heart were the vaso-dilators. Every one knows and appreciates the effect that the inhalation of the nitrite of amyl has upon the peripheral circulation. But although its evidence is so demonstrable, I am not at all sure how it acts beneficially upon the heart. The most obvious explanation is not necessarily the true one. I have already expressed my doubts as to the correctness of the explanation of its action in angina pectoris (§ 58). The usual explanation is that there is an arterial spasm which offers great resistance to the heart, and the amyl nitrite relieves the spasm. There can be no doubt but that the relief obtained by many people during attacks of angina pectoris where there is high blood-pressure by means of amyl nitrite or trinitrin, may be due to lowering of the blood-pressure, but there are other cases in which the drugs give relief where there is no arterial spasm, and if the arterial pressure be taken as the measure of the spasm, it will be found that a few minutes after the effect of the amyl nitrite has passed off the arterial pressure may be greatly raised.

The employment of other nitrites, as nitro-glycerine, to reduce permanently high arterial pressure, has been in my hands of very little avail,

though I have persevered with the drug for considerable periods. The action of the nitrites is, as a rule, transient, and they seem to have little lasting effect. I am now of opinion that in high pressures, with degenerated arteries and a diminished capillary field, they are useless. Even if they acted as permanent vaso-dilators, I doubt if their use would be justified in cases of high arterial pressure. Besides, one often notices that when by any means the high pressure is reduced, the patient is certainly no better, but often weaker.

In cases of high arterial pressure, with a good deal of discomfort, as pain and tightness across the chest, iodide of potassium has a great reputation, which has been justified in my experience. I have also used it with seemingly good results in elderly patients with recurrent attacks of bronchitis. At one time I thought it acted by lowering the pressure, but I found the good results occur with no alteration in the pressure. I have not used the large doses sometimes recommended, but 5 grains three or four times a day. These doses rarely produce iodism.

I have been struck with the good effects of chloral in some of these cases of high blood-pressure, given in small doses (5 grains) two or three times a day, as well as in larger doses to induce sleep. Many of my patients who suffered from severe attacks of angina pectoris found chloral gave them the greatest relief, and some used to carry it when liable to these attacks. The attacks did not come on with sudden violence, but gradually after exertion, and the chloral often gave complete relief in about ten minutes.

The vaso-constrictors have been of little use in my hands. I have repeatedly used adrenalin in cases of low arterial pressure, but it never produced any effect that led me to look upon it as of much value. A number of drugs apart from the digitalis group have a reputation as 'cardiac tonics', but I could never find any evidence of their effect upon the heart, beyond that indefinite beneficial tonic effect that follows the administration of such drugs as quinine. The most popular remedy of this class is strychnine, or some preparation of nux vomica. I have carefully sought for its special effect on the heart and found none. When I inquired into the evidence for its supposed good effect, I found that it was practically all clinical, and clinical evidence endows the drug with the most diverse properties. It is recommended as a cardiac stimulant in slow-acting hearts, and even in heart-block it is said to quicken the beat. It is also recommended in the too-excitable heart, as when there are extra-systoles, and in the rapid heart of acute myocardial affections. It is said to be beneficial in cases of low blood-pressure, and equally beneficial in cases of high tension, and even in angina pectoris. The evidence that can show a drug to possess the property

of exciting the sluggish and of soothing the excited, of raising the low pressure and relieving the high, speaks more for unreasoning faith in the drug than for the beneficial properties of the drug itself.

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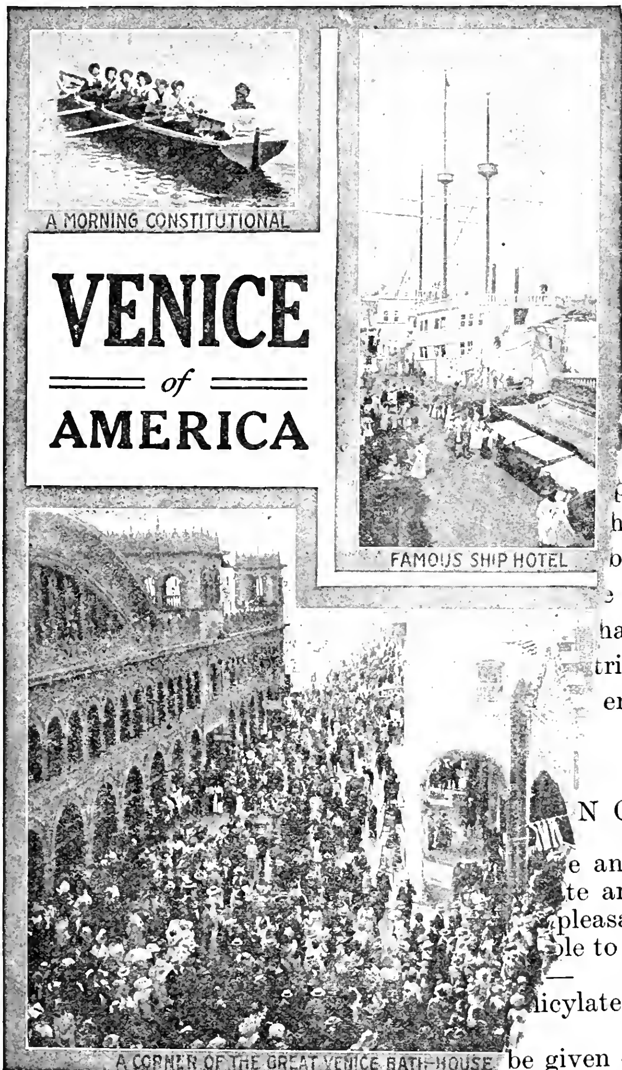

...N OF SODIUM SALICYLATE

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...le to give large doses, if the following

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...be given every two hours during the day
...night: ten doses in twenty-four hours.
...symptoms are produced, two or three doses
the administration should be recommenced,
one-half, or two-thirds of the previous dose,

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of exciting the sluggish and of soothing the excited, of raising the low pressure and relieving the high, speaks more for unreasoning faith in the drug than for the beneficial properties of the drug itself.

Since this was written, Professor W. E. Dixon [392], in an address before the section of Pharmacology and Therapeutics of the British Medical Association, said: 'Both these drugs (ether and strychnine) are still employed on the supposition that they excite the heart to increased activity. The effect of ether is to depress nerve-tissues; in very large doses it tends to depress muscle-tissue, including cardiac muscle, but it never excites. Strychnine likewise has no direct stimulant action on the heart; by exciting the vaso-motor centre it may slightly increase the vaso-motor activity indirectly, but it should never be put in the same category with digitalis, lead, and other cardiac drugs.'

The reason I dwell upon this is to warn against placing trust in drugs to the neglect of the essential requisite in treatment. There is a very common tendency to attribute the improvement in the heart, not to the rest that it has obtained, but to the special drug or method employed. This is like attributing the recovery of a famishing man who is restored by a plate of nourishing soup to the garlic with which the soup is flavoured.

In acute febrile conditions, when the heart is invaded by some organism, vaccine or serum therapy holds out some hope of effective treatment, but of this I have had no experience. In acute rheumatic affection of the heart, the use of the salicylates is often attended by great benefit. These should, however, be pushed, and, though I have not used them frequently in the manner recommended by Lees, yet I have seen such good results that I think Lees's method should be given a trial. He has kindly written out for me the directions and precautions he employs in giving large doses of salicylate of soda :—

NOTE ON THE ADMINISTRATION OF SODIUM SALICYLATE

For the effective treatment of acute and subacute rheumatism and of chorea, large doses of Sodium Salicylate are often necessary. In spite of the tendency of this drug to produce unpleasant symptoms, such as vomiting, deafness, &c., it is almost always possible to give large doses, if the following method of administration be adopted :—

(1) To every dose of Sodium Salicylate, twice as much Sodium Bicarbonate must be added.

(2) The combined drugs must be given every two hours during the day and every four hours during the night: ten doses in twenty-four hours.

(3) When any unpleasant symptoms are produced, two or three doses should be omitted. Then the administration should be recommenced, using smaller amounts; one-half, or two-thirds of the previous dose,

according to circumstances, but given as before ten times daily. The amount should then be increased as rapidly as possible.

(4) The initial dose may be 15 grains for an adult, 10 grains for a child of 8–12 years, 5 grains for a younger child. This amounts to—

150	grains	Sod.	Sal.	with	300	grains	Sod.	Bicarb. daily
100	,,	,,	,,	,,	200	,,	,,	,, ,,
50	,,	,,	,,	,,	100	,,	,,	,, ,,

(5) The amount of these drugs should be increased every day, or every second day, by 5 grains of Sod. Sal. and 10 grains of Sod. Bicarb. in each dose, or daily 50 and 100 grains respectively.

(6) The administration should be temporarily suspended if vomiting, deafness, tinnitus, or any tendency to delirium be produced. But the period of suspension should be short—rarely more than 12 hours—often a shorter period suffices if the amount given be reduced.

(7) The amount of Sodium Salicylate required in ordinary acute rheumatism is from 100 to 250 grains daily ; in chorea from 200 to 350 grains ; in chronic subacute rheumatism the dose may require to be raised to 450 grains or even 500 grains.

(8) Children require nearly as large doses as adults, for in them the rheumatic infection is specially virulent.

(9) Enough Sodium Bicarbonate must be employed *to render the urine alkaline.* If this be secured, and constipation be prevented, the Salicylate may be given freely without apprehension. But if a rheumatic child be costive and too little bicarbonate be given, it is possible to bring about a condition of poisoning by Salicylate—drowsiness, and deep breathing, increasing to air hunger and coma, with acetonuria and the odour of acetone in the breath. This is the real danger from Salicylate ; it may also be produced by aspirin. But it can always be avoided by attention to the directions given above. The existence of this danger should make the physician watchful, but it should not lead him to deprive the patient of the great benefit to be obtained from large doses of the drug.

§ **248. Oxygen.**—Although oxygen has been employed for many years in affections of the heart and lungs, it must be confessed that its employment has not been followed by the good results which were hoped for. Every one who has used it to any extent in affections of the heart has found it of distinct advantage in exceptional cases, but on the whole the results have been disappointing. In a measure it may be that this disappointment was due to two things, (1) that there are only certain cases suitable for this oxygen treatment, (2) that it has not been given in sufficient amount.

To take the second of these suggestions first, I was impressed with the observations of Hill and Flack[403], whose experimental work demonstrated that oxygen relieved the strain on the athlete's heart, and that by the somewhat open method usually employed there was not sufficient concentration of the oxygen inhaled. Thus Hill and Flack found that the

percentage of oxygen inhaled in the ordinary open method by a funnel was 19–27 per cent. in the alveolar air, whereas by the method described below there was over 70 per cent. of oxygen in the alveolar air.

In regard to the cases suitable for oxygen, slight temporary relief is obtained by all cases that are more or less cyanotic. I had hoped, however, that it would have proved useful in another class of case where the suffering from breathlessness and pain is often very distressing, such as in cardio-sclerosis with cardiac asthma, Cheyne-Stokes respiration, and angina pectoris. In these cases I reasoned that the exhaustion of the heart muscle that induced these distressing symptoms was due to deficient nourishment. As, however, the tissues were bathed in the lymph which contained the salts necessary for the nourishment of the heart, it might be the deficiency of oxygen that was the main cause of exhaustion. I therefore employed Hill's method of giving concentrated oxygen, and in the first few cases I met with most gratifying success: patients who had suffered for months from restless and disturbed nights due to dyspnoea and pain obtained great and, in some cases, immediate relief. Further experience, however, showed that apparently similar cases in which *prima facie* I would have expected the same satisfactory results, little or no relief was obtained. The matter therefore requires further consideration in order to recognize more accurately the class of patient that should benefit by oxygen, and I give here a short description of Hill's method, which I have used.

I have found that in attempting to give oxygen in a concentrated form, a mask like a chloroform mask conveys to the patient a sense of suffocation. Hill employs a mask of a very simple kind. A piece of light macintosh cloth about two feet square is taken, and near one side a hole is cut in the middle over which is glued a piece of transparent celluloid six inches square. An elastic band is attached around this border of the macintosh, so narrow that it will grasp the head lightly in such a position that the celluloid covers the face while the remainder of the macintosh is lightly packed around the neck. The oxygen direct from the cylinder is introduced by a tube through a hole below the celluloid. The cool current of oxygen is felt as refreshing, and is greatly appreciated by the patient. I generally give a fair stream of oxygen for 15–20 minutes—using in that time 10–12 feet of oxygen.

When this mask is not available (it can be had from Siebe, Gorman & Co., 187 Westminster Bridge Road, London) I have employed a lady's hat-box, cutting out of the side a space for the patient's neck, so that the patient's head goes comfortably into the box. I then put on the lid, and pass the tube from the cylinder through a small hole in the side of the box.

This method of giving the oxygen is rather wasteful, and for economy

another method may be tried where the oxygen is given by means of a bag, and the patient inspires and expires through a tube, where by a valvular arrangement the expired air passes through a tin containing sticks of caustic soda, which takes up the CO_2 while the oxygen is led back to the bag to be respired again. This apparatus is also made according to Hill's design by Siebe, Gorman & Co.

Leonard Hill has also devised a simple apparatus by which a patient can make a small quantity of oxygen and use it as desired. It consists of a small metal box—the generator—connected with a vulcanized rubber breathing-bag. The bag holds about 15 litres of gas, and ends in a mouth-piece. The bottom of the generator is formed by a screw-lid fitted with asbestos washer. A piece of wire gauze is interposed between the generator and the bag. A screw-clip closes the mouth-piece during the generation of the gas. The oxygen is generated out of oxylithe (Na_2O_3) by contact with water, and a solution of caustic soda results which is used to absorb the exhaled carbonic acid. $Na_2O_3 + H_2O = {}_2NaOH + O_2$. The oxylithe is sold in flat tins containing ten blocks to the pound, and three blocks make a charge sufficient for fifteen minutes' inhalation.

The bag is laid flat on the table and emptied of air. The oxylithe is then put into the generator, and water introduced into the bag through the mouth-piece. The screw-clip is then closed, and the bag raised. Some of the water then enters the generator, and the bag becomes full of oxygen. The patient puts the mouth-piece into his mouth and a soft clip on his nose, and, opening the screw-clip, breathes in and out of the bag, shaking the solution round the bottom of the bag meanwhile to absorb the carbonic acid exhaled. When about two-thirds of the bag of gas has been used up, the instrument is washed out and hung up to dry.

CHAPTER XXXIV

Treatment (*continued*)

§ 249. The action of digitalis on the human heart.—In inquiring into the therapeutic action of digitalis, I found that the result of clinical experience was that digitalis was undoubtedly beneficial, but that it was uncertain in its action, and that the various preparations varied much in their potency. A wide divergence of opinion existed in regard to what cases were suitable for its administration.

On starting this inquiry I was at first lost in confusion, unable to find any sure line to follow. I systematically gathered a great number of cases where I had given digitalis, and, on classifying the results obtained, certain clear issues came out, which gave very definite lines for me to follow in the subsequent investigation. A summary of these results I give here, dealing somewhat fully with a few illustrative cases in Appendix VI.

In regard to the question of preparation, I limited myself to three different forms—viz. B.P. Tincture of Digitalis, Nativelle's Digitalin Granules, and the pills containing Digitalis, Squills, and Calomel (℞. Digitalis pulv. gr. $\frac{1}{4}$, Scillae pulv. gr. $\frac{1}{4}$, Hyd. subchlor. gr. 1). I did not extend my observations to other forms, as it seemed better, with my limited field, to get a definite result from these very diverse preparations, than to arrive at an indefinite result with a multiplicity of preparations. The results of this investigation showed that all three forms were equally potent in certain cases and equally impotent in other cases. As a rule, when in one patient a certain definite reaction was obtained by one preparation it was also obtained by the exhibition of the others. I drew the conclusion that variability in the action of the drug did not depend so much on the preparation as on the nature of the lesion with which the heart was affected.

§ 250. **Action on dilatation of the heart.**—When I came to inquire into all the symptoms in these cases where digitalis had a beneficial action, I found them limited to those in which there was dilatation of the heart. But it was not equally effective in all cases of dilatation. In cases of old-standing rheumatic affection with dilatation it was generally very effective, and in many cases with no history of rheumatism; while in others of similar origin and with heart failure, but *without dilatation* (e.g. mitral stenosis), digitalis had no beneficial effect. In fact, I failed to see any evidences of a good result in any patient where the heart was not dilated.

The good results obtained by the use of digitalis are doubtless due to the specific action of the drug on the function of tonicity.

In acute dilatation of the heart, as in febrile affections, I could never find any improvement under it; nor was there much benefit in cases of dilatation secondary to advanced cardio-sclerosis. In a few cases some slight improvement might take place, especially if there was dropsy—a slight increase in the quantity of urine. But as a rule no appreciable benefit resulted.

§ 251. **Action on rate and on the nodal rhythm.**—When the rhythm was normal, i.e. when the ventricular contractions followed the auricular, digitalis did not often have much effect beyond slowing the rate to a slight extent. In a few cases of dilated heart with frequent action, due to old-standing rheumatic affection, the heart slowed under digitalis, but it never fell below the normal. In rapid hearts in febrile affections it never had any influence, nor in the quick heart of exhausting disease, as in tuberculosis, the reason probably being that a far more powerful poison was already in the possession of the heart.

In cases of nodal rhythm the action of digitalis is sometimes little short of miraculous. It is really to its action in these cases that the digitalis group of remedies owe their great reputation. I have endeavoured to find out in the literature of the subject the nature of the cases which have led authors to eulogize digitalis, but I have found the greatest difficulty. Most writers deal in such general terms that it is evident they have not realized the nature of the heart's lesion in which they found digitalis effective. Where particulars have been given, I could infer with fair certainty that the cases were those of rheumatic hearts with the nodal rhythm.

In Figs. 152, 153, and 154 are given tracings which show the action very typically. In Fig. 152 the heart is irregular in its action, 80 beats per minute. The jugular pulse is of the ventricular form (nodal rhythm), and is typical of the condition so common in advanced rheumatic hearts. The heart was dilated; there was a systolic and long diastolic murmur at the apex (see shading under Fig. 153), the urine was scanty, and there was

much dropsy. The patient was put upon the digitalis, squills, and calomel pill three times a day. In ten days' time the heart became much slower, and took on the rhythm shown in Figs. 153 and 154. The curious doubling

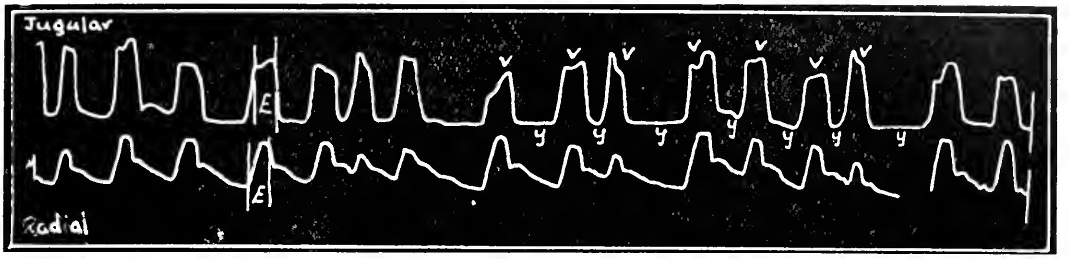


Fig. 152. Tracings from an old rheumatic case with the nodal rhythm, showing the characteristic irregularity and ventricular venous pulse. At the post-mortem examination there was both mitral and tricuspid stenosis. Before digitalis. (Figs. 153 and 154 are from the same patient.)

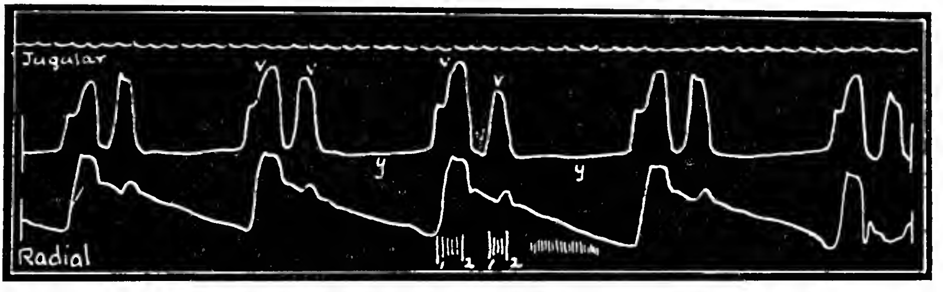


Fig. 153. Shows the characteristic effect of digitalis in old rheumatic hearts with the nodal rhythm. The figures 1, 2, represent the first and second sounds of the heart, and the shading represents the murmurs present.

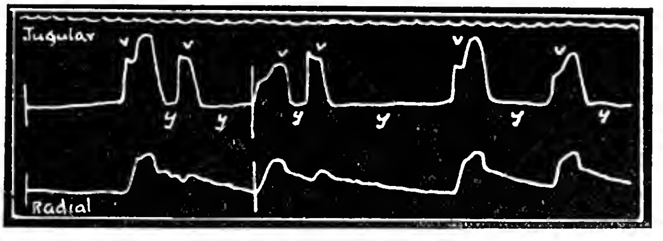


Fig. 154. Shows a digitalis effect with coupled beats and slow single beats.

of the beat shown in these tracings is a very common result of digitalis in this class of case. With the slowing of the pulse the patient's condition improved; the urine became more abundant, and the dropsy to a great extent disappeared.

The curious coupling of the beats in Fig. 153 requires consideration as a matter of practical importance and scientific interest. If cases were judged by the pulse alone the nature of the change would be entirely lost.

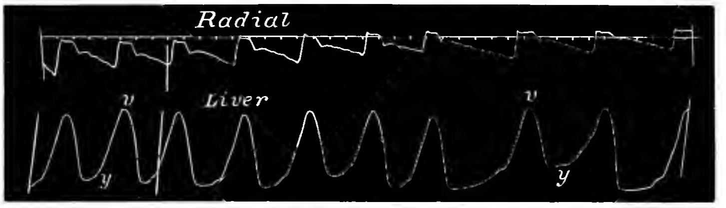


FIG. 155. Tracings of the radial and liver pulses before administration of digitalis from an old rheumatic heart, with great dilatation, and where the mitral valves were shrunken at the post-mortem examination. Figs. 153 and 154 were taken from the patient after the administration of digitalis.

Thus, Fig. 155 is from a patient with great dilatation of the heart and mitral incompetence—the valves damaged by an old rheumatic affection of the heart. Under digitalis the heart became slow, the radial pulse

FIG. 156. Tracing of the radial pulse (28 per m.) after digitalis. The real nature of the slowing is shown in Fig. 157.

being at the rate of 28 per minute (Fig. 156). Tracing Fig. 157, taken simultaneously from the apex and liver, showed that both ventricles contracted together, the apex giving the characteristic cardiogram of the left

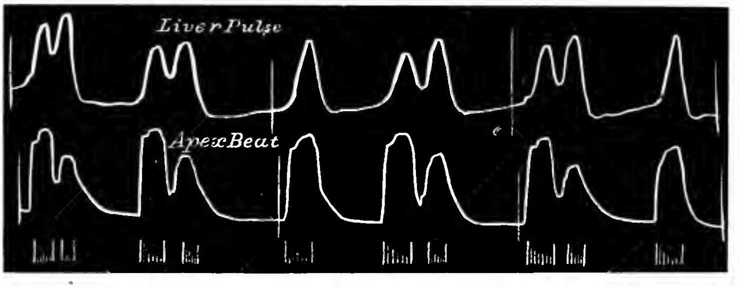


FIG. 157. Simultaneous tracings of the liver pulse and apex beat, showing complete harmony in the rhythm of both ventricles. The sounds and murmurs present are diagrammatically represented. After digitalis.

ventricle, while the liver pulse was produced by the right ventricle. The curious coupling of the beats is shown here, and the slow radial pulse (Fig. 156) is seen to be due to the fact that the smaller beats did not reach the radial.

The same features are brought out in the tracings from another patient. Fig. 158 shows the jugular and radial pulses when the patient was not under the influence of digitalis. Figs. 159 and 160 show the action of the

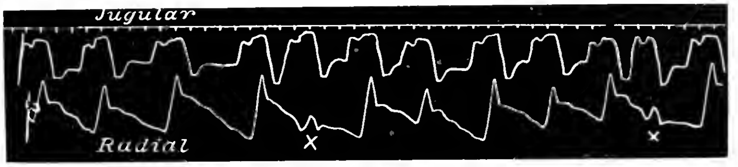


Fig. 158. Simultaneous tracings of the jugular and radial pulses. The jugular pulse is of the ventricular type, and the tracing shows complete agreement in rhythm between the right and left ventricles. From an old rheumatic heart, in which, at the post-mortem examination, there was found great stenosis of the mitral orifice. Before digitalis. Figs. 159 and 160 are from the same patient.

heart due to digitalis. In these tracings, the one of the apex and radial and the other of the liver and radial, the characteristic coupling of the beats seen in the apex and liver is not observed in the radial because the smaller

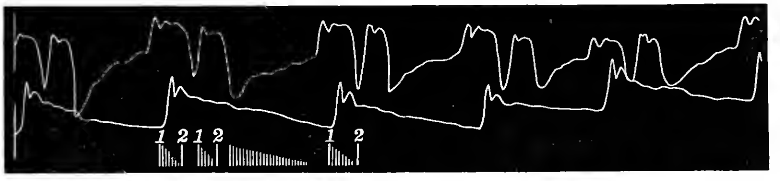


Fig. 159. Simultaneous tracings of the apex beat and of the radial pulse from a case of mitral stenosis. The coupled beats are well marked in the apex tracing. The shading underneath shows the time of the murmurs. After digitalis.

beats did not affect the radial pulse. In this case there was a long diastolic murmur, as shown by the shading, and the post-mortem examination showed extreme stenosis of the mitral orifice.

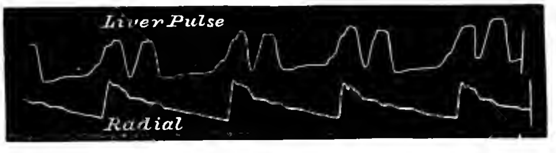


Fig. 160. The coupled beats are well marked in the liver tracing. After digitalis.

I have a large collection of tracings showing the same results, and every one of them came from patients with the nodal rhythm, and with a past history of rheumatism. The practical importance of this reaction lies in the fact that the appearance of these coupled beats is at once the signal to lessen

the dose of digitalis. No good is to be obtained by pushing it further, and when one can just manage to keep the patient at the stage where this tendency occurs the best results from digitalis will be obtained—the patient feels easier, makes more urine, and the dropsy is kept under control.

The scientific interest lies in the fact that no other form of heart ailment produces this reaction. It has never been produced experimentally, and cannot occur while the auricles are active and occupying their normal place in the cardiac cycle ; in other words, it is a reaction limited to patients with the nodal rhythm. I have thought much on the nature of this rhythm, but my conclusions are of too speculative a nature to be of any real value. I wish, however, to point out that the second of the linked beats are not extra-systoles. In § 141 I defined an extra-systole as the premature occurrence of an auricular or ventricular systole while the fundamental or sinus rhythm of the heart was maintained—the retention of that rhythm accounting for the character of the irregularity. Here there is no sinus or fundamental rhythm, and as a consequence the pauses between the beats are continuously liable to variation.

In cases with the auricles active, the production of extra-systoles by digitalis does occur in a small proportion of cases, but usually only after the drug has been pushed rather far. In Case 25, Appendix VI, this coupling of the beats occurred while the patient had the nodal rhythm. After a time the normal rhythm was restored, and when digitalis was pushed extra-systoles occurred, but the whole character of the heart's action was very different, as a study of the tracings given from this case will show. This coupling of the beats has been noticed by other observers. Thus Broadbent[2] describes the coupled beats as a result of digitalis, but he and others have failed to appreciate the class of case in which it occurred, and did not realize it was a reaction peculiar to this kind of heart.

The manner in which the heart-rate can be played upon by digitalis in these cases of nodal rhythm is very remarkable. I have frequently seen the heart increase in frequency, sometimes very greatly (up to 120–130 beats per minute) when the digitalis was withheld, and in the course of a few days digitalis often soon slowed it down to between 60 and 70 beats per minute. (See Case 25, Appendix VI.)

But the effect is not only upon the rate of the heart : the size of the heart diminishes, the urine increases, and the dropsy disappears. If there be much liver enlargement, the digitalis at the same time diminishes the size. Fig. 66, p. 127, was taken from a pulsating liver at the level of the umbilicus. The patient was at the time extremely breathless, and had to

be propped up in bed. Tincture digitalis, 15 ♏ three times a day, was prescribed, and in three days' time he was up and about, and no sign of the liver could be detected below the ribs.

I am convinced that it is the striking effect of digitalis in these cases that has led clinicians to form such a high opinion of this drug. Failing to recognize that the action depended on the nature of the lesion, they have sought to find a similar benefit in cases not so susceptible to it, and from this have arisen the confusion and contradictory statements in regard to the action of digitalis.

No such reaction as that shown in Figs. 152–160 can be obtained, unless where the nodal rhythm is present. And even amongst cases of nodal rhythm a further differentiation has to be made, for I never get this result in cases of nodal rhythm secondary to cardio-sclerosis associated with arterial degeneration. In these cases I have sometimes seen a little improvement—an increase in the flow of urine and a diminution of the dropsy, and it may be a slight slowing of the heart's rate—but never anything approaching the slowing in old rheumatic cases. In many cases, though I have pushed the drug till the patient was sick and had diarrhoea, I have seen no alteration in the heart's condition. This difference is probably due to the greater extent of the degeneration of the heart muscle.

§ 252. **Action on conductivity (heart-block).**—In a great many cases I have been able to produce a mild form of heart-block by the administration of digitalis, and I can now recognize the class of case in which this occurs. I have already pointed out that the interval between the auricular systole and the ventricular can be determined by the length of the *a–c* interval in tracings from the jugular pulse (§ 163). If this interval be above one-fifth of a second (unless in a very slow heart) it is due to a depression of the function of conductivity of the a.-v. bundle. When the heart's action is over 100, with the interval one-fifth of a second, a depression of conductivity may be suspected. By the administration of digitalis in a great number of my patients I have been able to increase this depression of conductivity, so that ventricular systoles have dropped out because the stimulus from the auricle did not reach the ventricle.

In Fig. 161 there is a wide *a–c* interval (two-fifths of a second). I gave the patient from whom this tracing was taken three drachms of tincture of digitalis in the course of four days, and her pulse became irregular, due to the dropping out of ventricular systoles (Fig. 162). Some years previously I had produced a similar effect in this patient by digitalis, when every second ventricular systole dropped out (Fig. 163).

When digitalis is prescribed for a patient with a regular pulse and the

heart becomes irregular, it is probably due to this cause. The nature of the irregularity can be inferred with fair certainty by auscultation when the heart sounds are found to be absent during the pause. Extra-systoles sometimes follow the administration of digitalis, and the irregularity of the pulse may resemble the dropping out of beats due to heart-block, but with

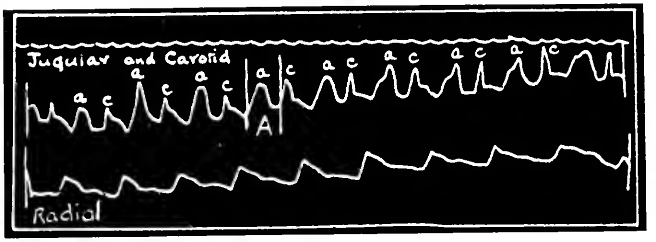


FIG. 161. The *a–c* interval is twice the normal period (two-fifths of a second in place of one-fifth), indicating a delay in the stimulus passing from the auricle to the ventricle. (This and the next two tracings are from a female, aged 24 years, suffering from mitral stenosis.)

extra-systoles the short, sharp sounds of the heart during the pause in the radial pulse will usually be detected (Fig. 87). In many cases where there is dilatation of the heart, as soon as the irregularity appears the patient experiences a good deal of relief, though sometimes the throb after the pause is distressing. (See also Cases 26 and 27, Appendix VI.)

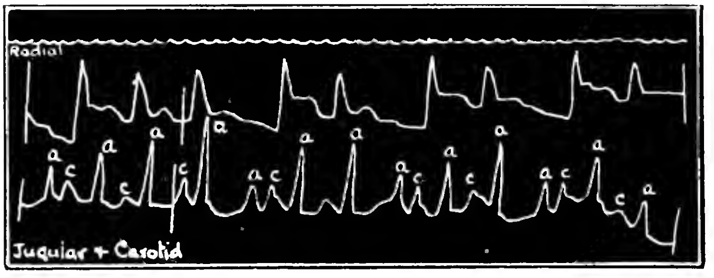


FIG. 162 shows the missing of a ventricular systole at frequent intervals on account of the delay in the stimulus passing from the auricle. It will be noticed that the wave *a* is perfectly regular in its appearance, and that its relationship to the carotid and radial is variable (digitalis effect). Note the gradual increase of the size of the auricular wave, *a*, before the intermission. This is due to the gradual increase in the width of the *a–c* interval until the auricular contraction occurs before the preceding ventricular systole is completed (see also Fig. 114).

In most of the cases in which I have been able to produce this condition of mild heart-block there has been a previous history of rheumatic fever.

§ 253. **Action on contractility.**—If tracings be taken systematically of patients under the influence of digitalis, evidence of a depression of contractility may occasionally be detected in the form of the pulsus

alternans. It is generally so slight as to escape detection unless a tracing be taken.

In Figs. 164 and 166 are tracings of the pulsus alternans, due in the one

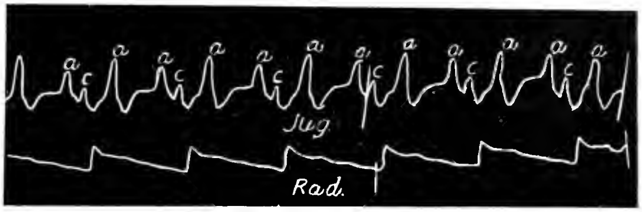


FIG. 163. Slowing of the pulse due to digitalis depressing the conductivity, so that the ventricle responds only to every second stimulus. While the ventricle contracted forty-eight times per minute the auricle contracted ninety-six times.

case to digitalis, squill, and calomel pill, and in the other to the tincture of digitalis. Curiously enough, the appearance of this change in the rhythm of the pulse was accompanied by an improvement in the patient's con-

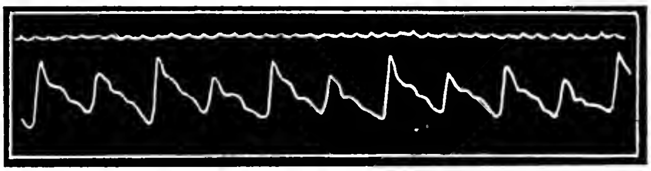


FIG. 164. Typical pulsus alternans due to digitalis. (Case 9, Appendix II.)

dition. The reason for the improved condition when the conductivity of the a.-v. bundle and the contractility of the heart muscle was depressed, is in all probability a coincident improvement in the tonicity. This view

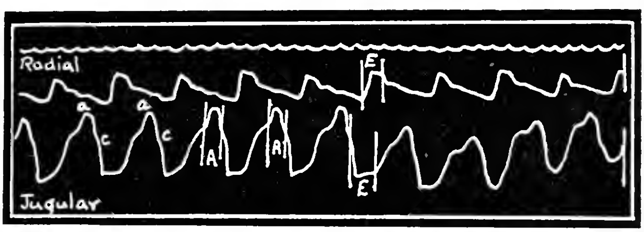


FIG. 165. Taken at the same visit as 164, and from the same patient, to show that the conductivity was not affected—the *a–c* interval being normal (one-fifth of a second).

is confirmed by some observations Gossage has shown me. That the function of conductivity was unaffected in these two cases is shown by the normal *a–c* interval in the jugular tracings (Figs. 165 and 167).

§ 254. **Action on blood-pressure.**—Contrary to expectation, I found that it is only in exceptional cases that the administration of digitalis

raises the blood-pressure. I have repeatedly pushed it until distinct evidence of its physiological action was produced on the stomach or on the heart, without any apparent rise in the blood-pressure. Even when the patient was greatly benefited by the drug, I have frequently seen little or no evidence of an increase in the blood-pressure. The only cases where I did find increase in the blood-pressure were in extreme dilatation of the heart with

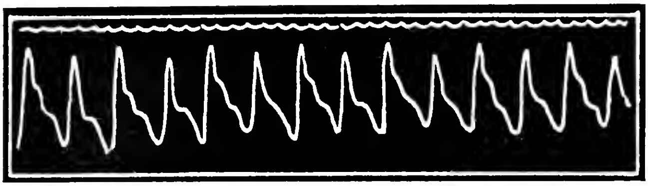


FIG. 166. Typical pulsus alternans due to digitalis.

considerable dropsy. In these cases a slight rise in the blood-pressure accompanies the improved condition under digitalis.

§ 255. **Digitalis in practice.**—In the following summary of the conditions in which digitalis will be found to act beneficially it may seem that its field of usefulness is far too restricted. While I do not think so, I wish to insist upon the limitations of its usefulness for a very important reason,

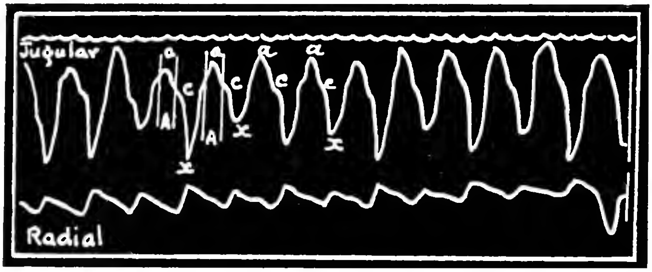


FIG. 167. Taken at the same visit as 166, and from the same patient, to show there was no affection of the conductivity, the *a–c* interval being normal.

namely, because the reliance placed upon this drug has led to the belief that it should be tried in all forms of heart affection, with the result that valuable time which should have been spent in putting the heart under favourable conditions has been wasted.

The condition of heart failure where it acts best is where there is dilatation of the heart with healthy muscle-fibres that have been exhausted in the endeavour to overcome abnormal resistance. This effect is best seen in old rheumatic affections of the heart, but it may also be observed in other cases with a fair amount of healthy muscle. It is of very little use

when the dilatation of the heart is due to extensive degeneration of the muscle-fibres, as in cardio-sclerosis. In a sense the degree of reaction gives a measure of the extent of the degeneration—the greater the extent of the degeneration the less the reaction. It will be found to be of little value when the heart is already in the grip of some poison, whether it be the specific organism of such disease as rheumatic fever, pneumonia, or the toxins of such diseases, or such poisons as alcohol and arsenic.

This conclusion, drawn from the careful study of individual patients, is in striking accordance with the conclusions drawn by Cushny[94] from experiment: 'In cases of dilatation of the heart with a weak and insufficient systole its action is almost specific. This is true whether one or both ventricular chambers are affected, so long as the cardiac muscle has not undergone degeneration.'

When the heart becomes continuously irregular (nodal rhythm) in old rheumatic cases, digitalis usually acts with great promptness and certainty. In these cases the administration should be kept up for considerable periods, just sufficient of the drug being given to keep the rate between 60 and 70 beats per minute, or until the characteristic coupled beats appear. Sometimes good results are obtained when the rate cannot be reduced so low. Under such circumstances the condition of the patient in other respects should be the guide, the dose being modified according to the degree of improvement.

In cases of nodal rhythm due to other conditions the heart is not so susceptible, and great caution should be exercised in the prescription of digitalis here. Occasionally we read of precautions in the use of digitalis, and of its tendency to cause a fatal stoppage of the heart, but I have been unable to determine the form of heart disease in which these sudden deaths are liable to occur. The only cases of sudden death I have seen during the administration of digitalis were unyielding cases of nodal rhythm. In one patient with cardio-sclerosis I was carefully pushing it, when the patient died suddenly in the night. In another case of rheumatic origin, digitalis had little effect, and the patient had taken only an occasional dose when she died suddenly while dressing one morning. But I am not clear in my mind that the digitalis had any part in the sudden end of these cases, and there was no preliminary slowing of the heart's action.

§ **256. Other drugs of the digitalis group (strophanthus, squills, helleborein).**—I have used other drugs of this group (strophanthus, squills, helleborein), but only to a limited extent. So far as my observations extended, I found that if digitalis failed to act they also failed. I tried strophanthus on several cases that were susceptible to digitalis and got

the same reaction, but only after a much longer time. This result was in striking agreement with the experience of a very intelligent man whom I saw in consultation. He had had rheumatic fever, and his heart had acquired the nodal rhythm. For many years he had been liable to frequent breakdowns, but could pull himself together with digitalis, and he always knew the exact quantity to take. He told me he had tried strophanthus, but that, while it restored him, it took a much longer time to do so than digitalis.

Some observers have found that cases have responded to strophanthus after digitalis had been tried and been found ineffective. We must be on our guard against the conclusion drawn from such an observation, for it is possible that the digitalis had in a measure prepared the way for the strophanthus, for it often happens that no effect is obtained from the digitalis until it has been pushed for some time.

CHAPTER XXXV

TREATMENT (*continued*)

THERE are a great number of other methods that are useful in relieving the patient or in assisting in restoring the reserve force of the heart, and I deal here with the most important of them.

§ 257. **Venesection.**—In a number of cases the abstraction of blood from the patient affords very considerable relief. Unfortunately the relief is only temporary, and in extreme cases only delays the end. Although I have practised venesection in a great variety of cases, I cannot say I have seen it do any lasting good. The indications for its use that have been my guide have been distress in breathing, on account of great distension of the right heart. In cases of mitral disease this has generally been recognized by the increase of the heart's dullness to the right. In cases of high blood-pressure (cardio-sclerosis) it has sometimes been difficult to detect much enlargement of the right heart, and the tense filling of the veins of the arm has been the indication. I have always bled at the usual place—at the bend of the elbow—and abstracted from twenty to thirty ounces of blood. The immediate relief given to the patient is often very striking.

§ 258. **Exercises.**—The heart, like every other organ, becomes more efficient with reasonable exercise of its functions, while with a too limited exercise the store of reserve force gradually dwindles, so that most people who pursue sedentary occupations have a limited field of cardiac response. When such people exhibit some cardiac abnormality, such as an irregularity, a murmur, or a fainting attack, the easily exhausted store of reserve force shown by the shortness of breath and palpitation on exertion is too often taken as an evidence of serious affection of the heart. This is more

particularly the case in the young with sinus arrhythmia (youthful form of irregularity) and in chlorotic females. Improvement in all these cases is best obtained by the gradual increase in the daily amount of bodily exercise. In the great majority of cases of serious heart-failure, even after recovery has set in, the judicious employment of muscular exertion is beneficial. It may be a matter of difficulty to determine whether more serious cases are fit for exertion, and if so to what extent. There is a very simple rule that I have been accustomed to follow for many years with the greatest satisfaction: let the patient employ that form of muscular exercise which he can best do without cardiac discomfort, and never indulge in it after the first sign of discomfort. By discomfort I mean the various signs which are given by the heart when its reserve force is exhausted—as breathlessness, palpitation, sense of exhaustion, pain. Discomfort may be experienced first in the muscles exercised, when some particular group of muscles is more particularly employed, as certain thigh muscles in climbing, and certain arm muscles in playing golf—indications more of want of training of these muscles than of heart exhaustion. This form of discomfort need not prevent further exercise.

If the rule, that exercise should stop short of exhaustion, be followed, it is surprising what an amount of effort can ultimately be endured by patients who may once have suffered from extreme heart failure. This will be appreciated by those who practise among the classes employed in hard manual labour. They may have continually irregular hearts (nodal rhythm), with aortic and mitral murmurs, and may have suffered at times from the most extreme failure of the heart with extensive dropsy, yet they can undertake the most severe form of manual labour with no discomfort. In such cases the heart muscle is healthy and capable of acquiring a considerable store of reserve, and it is from observing such patients that I base the prognosis of heart cases so much on the power of the heart to regain its reserve force.

When patients can go out, their exercise should be in the open air, even though it is limited to certain gymnastic movements. If they can walk quietly, that in itself may be sufficient, and if the walk be taken systematically a great amount of reserve force may ultimately be acquired. As a rule, people benefit more by exercise when it has an object beyond the medical needs. Hence the added interest of a game or the study of objects of interest, as architecture, botany, &c., will add materially to the efficiency of the exercise. The particular taste of each patient has therefore to be studied, and the form of exercise prescribed that is likely to combine the therapeutic with personal interest.

When patients are confined to the house, or to bed, moderate exercise of the muscles proves useful, so long as it does not embarrass the heart. To this end the various movements and gymnastics may be of use.

The good results of exercise are obtained by a variety of obscure processes. The increased functional activity of the heart assists in accelerating its own circulation and in restoring its strength, but there are also changes in the circulation of the muscles exercised which lead to a freer flow of blood through the system. The contraction of a muscle expels blood from its capillaries, and is at once followed by a dilatation of the capillaries. This is well seen when the hand and arm are placed in a plethysmograph, and the variation in their bulk registered. After a single closing of the fist, the bulk of the arm at once increases from the dilatation of the capillaries of the used muscles.

§ 259. **Massage.**—A benefit similar to that of exercise can, in a measure, be attained by massage. It is not necessary that a skilled person should apply it, for that would exclude its use among the majority of sufferers. The gentle but firm intermittent pressure upon the muscles in the body is quite sufficient. In dropsical cases the gentle but firm massage of the legs may prevent the dropsy reaching the extreme degree where the skin becomes thin and glistening and liable at any moment to ulcerate. In some extreme forms of the condition I have referred to as the X disease, when there is a persistent contraction of the smaller blood-vessels of the extremities, massage has been attended with benefit.

§ 260. **Special movements and exercises.**—There is a number of methods employed involving muscular exercises that have gained considerable repute. Such methods may have a limited usefulness when patients cannot take natural exercise. Their chief recommendation seems to be in relieving the tedium of convalescence, and in giving mental satisfaction to the patient that something is being done. I have made a careful inquiry into the effects of passive resistance movements and voluntary contractions of the muscles, and could find no appreciable effect upon the heart. In certain people, especially those of a slightly neurotic habit, the slowing of the pulse at the end of the seance was sometimes very marked, but I found I could produce exactly the same result by employing, with equal solemnity, indifferent acts, such as stroking the finger nails and the shin bones. No effect was produced on hearts acting at a frequent rate in consequence of serious heart failure or the nodal rhythm. On no occasion could I detect any decrease in the size of the heart as a result of the movements. That certain hearts may become

slow and diminish in size after a month's restful treatment every one will acknowledge, but it is assuming too much to say that such results were due to the special method employed.

§ 261. **Baths.**—A very powerful influence can be exercised on the circulation by the immersion of the body in water; this may act in several ways, perhaps mainly depending on the temperature. Great therapeutic efficacy is claimed for certain waters, but it is very doubtful if the ingredients in these waters have any effect upon the heart, beyond their effect in stimulating the skin. My personal experience has been limited to observing the results in patients who have returned from the various spas, and I have seen nothing of their good effects to lead me to place hydrotherapy very high as a means of treating affections of the heart. The best results I have seen have been in patients who have bathed in the open sea. When I have had patients with heart trouble who were fond of sea-bathing, I have allowed them to indulge in it, warning them to be honest with themselves, and refrain if it brought on any sense of discomfort. In many cases the result has been extremely satisfactory, the whole system of the patients has been braced up, and they have returned from the holiday greatly benefited.

§ 262. **Spa treatment.**—Sea-bathing has, after all, only a limited sphere of usefulness, and many patients obtain great benefits from visiting spas, and the supporters of each claim for its waters some special virtue. In order to assess the value of these claims, it is well to bear in mind by what process benefit is obtained at the various spas. The vast majority of patients go there as much for a holiday as for treatment, and when a patient is sent there, it is often because the individual, in addition to his complaint, has been busy with his affairs, and his heart complaint has been thereby aggravated; or a patient is convalescent, and a change of air, scene, and mode of life is often found beneficial. As the various spas cater for the more enjoyable side of existence, they attract large numbers of invalids who naturally desire the reputed benefits of the waters, and drink them enthusiastically, or, if they cannot drink them, at the least bathe in them. It will thus be seen that the benefits gained at such places arise from a variety of sources, and it is but human nature to attribute what benefit has accrued to the factors that most appeal to the imagination, such as hot gaseous waters from the bowels of the earth. Every practitioner of experience will agree with me that a large proportion of heart cases return from their holiday greatly improved, and this improvement is not limited to those who went to some particular spa, but includes all sorts of places—spas, seaside and mountain resorts, sailing on sea and lake. It is evident

that results thus obtained cannot be due to the peculiar constituents or the waters of any single place.

§ 263. **The Nauheim baths.**—When I began to write this book the purpose was to give a faithful account of my own experience. It was no part of my project to enter into controversial matters, and in matters of dispute I have simply expressed my own views. But I feel it would be misleading if I passed in silence a method of treatment that has obtained a world-wide reputation which I consider out of proportion to its merits. Though I enter into this matter reluctantly, I conceive it none the less a duty to give my views on it, particularly as I am impressed with the injury done to individual patients through the unmerited reputation of the Nauheim baths among the medical profession. Institutes have been started for the financial exploitation of the Nauheim waters, and I must confess to a feeling of shame for my profession, when I consider the manner in which it has been imposed upon. One reads in sober English medical journals accounts of cures effected that seem like the puffs of an empiric remedy. One writer will tell how a patient obtained no benefit from his treatment, but was cured by a visit to Nauheim. Another describes how he watched the patients enter into the bath-room feeble, tottering, and livid, and how they came out upright and brisk, with a glow of health on their countenances. It is little wonder that the stay-at-home practitioner is impressed by all this dithyrambic praise. The following painful experience resulted directly from this indiscriminate exaltation of the virtues of the Nauheim waters. I saw a man in consultation whose history was this: He was seized with symptoms of heart failure, and not improving as he liked, his doctor advised him to go to Nauheim. An eminent physician was consulted later who also strongly recommended Nauheim. Visiting another part of the country, he was taken ill, and the doctor who saw him there also told him to go to Nauheim. He was so impressed with the advice given independently by three doctors, that he made up his mind to follow it and go to Nauheim. His circumstances were such that he had to stop all his professional work and to expend a sum of money that he could ill afford. He was only able to travel to Nauheim by easy stages, and took three days on the journey, arriving there spent and exhausted. He was put through the routine treatment of the baths, and had digitalis prescribed in addition. He returned to England worse than when he set out, though bearing with him a letter from the Nauheim physician saying that he had greatly improved by his visit. The patient himself shrewdly remarked that, seeing that he arrived there dead beat from his journey, it would have been surprising if he had not picked up a little by the rest, but as to his condition he had

gained no benefit, but the reverse, from his trip. When I saw him after his return, his was an undoubted case of advanced cardio-sclerosis, with extreme exhaustion of the heart muscle. The organic changes were irremediable, but the exertion of going to and from Nauheim had injuriously exhausted the heart, and no doubt hastened the end of the patient, in addition to exhausting his financial resources, for which those dependent on him had to suffer.

This is by no means an exceptional instance, and one physician of experience tells me that every year he is called upon to treat a number of the 'Nauheim wrecks', as he calls them, on their return. But I do not wish to seem to condemn a method without reason, and shall briefly recount my experiences in an attempt to appreciate the curative virtues of the Nauheim methods.

On arriving at Nauheim and interviewing several doctors as to how the efficacy of the cure was to be investigated, I discovered that in serious cases no practising physician believed the waters to possess sufficient curative properties, but that accessory means had to be taken if a good result was to be obtained. Nor could I find among those practising there any agreement in regard to what was the best accessory means. One said the waters were good when assisted by the additional movements attained by the machinery of the Zander Institute; another derided the use of the Zander machinery, and said the best effects were obtained from the baths combined with his specially invented method of exercise; while a third said the methods of the other two were of little avail, and that the best results were obtained when to the baths something extra was added—such as an electric current. When all these methods and baths were of little avail, every doctor prescribed in addition drugs of the digitalis group. It was hopeless for me to attempt to find out the efficacy of any given bath or method when such complications were introduced, so I did what little I could to understand the influence of the baths.

I found that ten to twenty years ago, when the notion was prevalent that to have a good heart you must have a strong pulse, these baths had a remarkable effect in strengthening the pulse, raising the arterial pressure 20, 30, and 40 mm. Hg. But nowadays the fashion being to soften a strong pulse, these waters are discovered to have a remarkable effect in lowering the arterial pressure. So remarkable are these waters that it is claimed that they can increase the pressure when it is low, and lower the pressure when it is high.

I found that these baths were so modified as to be of different strengths, and it was stated that the different baths were given according to the nature

of the complaint. But I could find no evidence of any rule being followed. I found that people with nothing the matter with their hearts were having the same baths as those who were suffering from severe heart affection. I also found patients with a weak frequent pulse having the same baths as others with a slow hard pulse.

I saw nothing which, by the greatest stretch of imagination, could confirm the statement that patients are to be seen entering these baths bent and ill and coming out of them straight and strong. In the patients I watched in the baths, I could discover no improvement from the single immersion. Certain effects on the heart, such as slowing of its action, did occur in several cases, notably in healthy hearts, as in my own case and in that of a friend whom I watched. This was in the strong sprudel bath, when the temperature of the water was 89° F. But it seemed to me merely a temperature effect, and this was confirmed by the fact that when I returned home I found my pulse-rate and that of my friend slowed in exactly the same manner when we lay in a bath of ordinary tap-water at the same temperature. I found this experience corroborated in a series of careful observations by Reissner and Grote, who compared the effects of the waters from these springs with those of plain water at the same temperature, and found the slowing of the heart entirely dependent on the temperature. This effect of temperature is practically never referred to, but is attributed to some specific effect of the waters on the skin. Thus in lying in the bath, the water being charged with carbonic acid, this gas comes off in innumerable small bubbles which can be seen adhering to the skin. At the same time the skin becomes red. These very simple phenomena are pointed out as in some way bringing about a reflex stimulation of the heart.

§ 264. **Cause of the efficacy of the spa treatment.**—It may be said, and truthfully, that large numbers of people flock to Nauheim and many of them derive great benefit from the treatment. I recognize this, and have carefully endeavoured to find out the reason for the success of the Nauheim methods. When the cases that are cured and the cause of their cure are strictly analysed, it will be found that at Nauheim what I call the essentials of treatment are carried out in an excellent manner. Everything is conducive to the restfulness of the patient. It is a pleasant place, sunny and well shaded, with beautiful gardens and an excellent band. People jaded with their cares and duties find here that repose which is essential to the recovery of the heart. A very large proportion of them are somewhat neurotic, and there is consequently a very susceptible mental element that can be influenced. The patient comes to Nauheim buoyed up with the reputation of the place. When he consults a doctor,

he is confidently told that the treatment will do him good—at once half the cure is effected in a great proportion of the cases.

Of wonderful cures I saw none. Pursuing my work in a remote manufacturing town, when I read of the wonderful cures performed at places like Nauheim, I imagined that these would be the class of cases that I failed to benefit. What was my surprise to find at Nauheim that the so-called wonderful cures that were being effected were identical with those that practitioners achieve at home.

I found at Nauheim that which I had also found at other spas, that the practitioners there were scarcely aware of what the human heart was capable. Those who, like myself, have practised largely among the better working class know what enormous capacity for recovery it possesses. Many of the ailments I saw at Nauheim would not keep a working man or woman from their work, and here they were going through elaborate methods of cure. I may frankly confess that I saw no patient get benefit at Nauheim who would not have done equally well elsewhere.

The argument is used that cases that have been treated elsewhere without success have obtained benefit at Nauheim. What doctor of experience has not the same to tell? I have repeatedly had patients place themselves under my care who had been treated by other doctors, and they have benefited. But I trust I am not so foolish as to fancy the recovery was due to my skill. In many heart cases the early stages of recovery are very protracted, and marked improvement often takes place with some slight change in the treatment, and the conclusion is often too hastily drawn that the recent change effected the cure, whereas the heart's power was being slowly restored by the treatment previously employed.

I have gone into the subject of the Nauheim treatment at length, so that the reader may appreciate the strength or weakness of the position I take up, and I want each practitioner seriously to consider his responsibility in every case before recommending an elaborate and expensive treatment. If the individual is well to do, and there is not much the matter with him—well, Nauheim is as good a place to send him to as any other. But when it means crippling a man's resources either by the outlay or by stopping his work, a grave responsibility rests upon his adviser.

In the case of growing boys and girls, I think Nauheim and the various methods are distinctly detrimental when the heart's weakness is purely functional and the symptoms consist in occasional fainting and some enlargement and irregularity of the heart. This class of patient is often sent there, and, in consequence of the elaborate ritual, they get the notion there must be something serious, and go through life under the impression

that they have a weak heart, with the consequences seen in the *malade imaginaire.* I have seen numbers of these going through these elaborate methods whom I would have sent out to the play-fields.

The assembling in crowds of neurotic people is a bad feature. They are so fond of detailing their symptoms to one another that they cultivate the habit of self-analysis. If this were done sanely, good might result, but it often ends in making the individual too self-conscious of what little infirmity he suffers from, so that I prefer to send my heart cases with a nervous element where they will associate as much as possible with healthy people, whose pursuits and tastes do not lean towards introspection.

APPENDIX I

The Pulse in Angina Pectoris

The condition of the arterial system immediately before and during an attack of angina pectoris has been the subject of a good deal of speculation, and a good many hypotheses in regard to diagnosis and treatment have been based upon the conditions that are supposed to exist. Unfortunately, very few observations have been made to determine the actual condition, and those that have been published do not bear a very critical examination. It is only at unexpected times that one has the opportunity for making the observation, and the circumstances are rarely suited for making these sufficiently accurate. I have been somewhat fortunately circumstanced for this purpose. For many years while I was in Burnley I had been consulted by large numbers of people with affections of the heart. Three of the four roads that led past my house were steep, and the patients suffered in climbing the hill. Not infrequently in my consulting room, or in my waiting room, patients would be seized with attacks of angina pectoris, and I have had many times to administer amyl nitrite for their relief. As I always had my sphygmograph ready, I frequently obtained very good tracings. I may say that in not a single case did I ever find any evidence of vascular spasm, recognizable by the finger, nor had I any difficulty in getting tracings on account of the smallness of the pulse. While one cannot always infer from the size of the tracing that the radial artery was large, yet it does offer confirmatory evidence. A small deformed tracing may easily be got from a large radial pulse, by hasty and inaccurate adjustment of the instrument, but it is rare to get a large tracing from a small contracted artery, and I give the following tracings merely as corroborating the sensations imparted to my finger.

In tracings taken before and after the administration of amyl nitrite, the size of the artery and the character of the tracing showed very little difference. During the administration there was always the characteristic changes—increased rapidity and lowering of the dicrotic notch, as in Fig. 169. It may be said that the attacks in the cases cited were after bodily exertion had exhausted the heart, and that the attacks that come on when

the patient is at rest may be due to arterial spasm. I have also had occasion to see a good number of cases who suffered from attacks while in bed, and here likewise I detected no arterial spasm. In a few cases I have found the arterial pulse small during an attack, and in one case imperceptible ; but these were cases of extreme exhaustion of the heart, with very feeble heart sounds, and the administration of amyl nitrite had no effect. Two of these patients died a few hours after my examination.

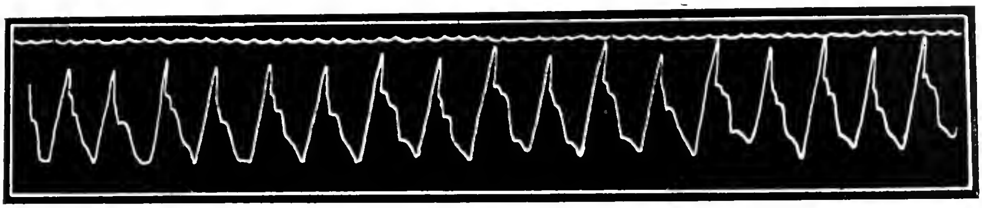


Fig. 168. Large alternating pulse during an attack of angina pectoris. B.P. 190. (Case 1. Figs. 169 and 170 are from the same patient.)

I have a large amount of material to select from, but I limit myself to a few cases of the most severe type.

Case 1.—Male, aged 52 (whose case is described more fully later—Case 23, Appendix V). Had suffered two years from breathlessness. As he had a well-marked pulsus alternans I have made a large number of observations on him. His condition fluctuated very much. During a period when he was worse than usual he called to see me, walking up a steep hill on

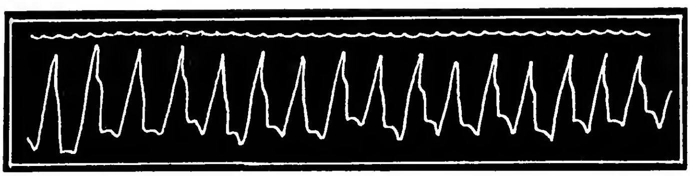


Fig. 169. Under amyl nitrite. (Case 1.)

the way. He began to feel a tightness across his chest which developed into a severe pain. I examined him and took a tracing of his pulse (Fig. 168). I took his blood-pressure and found it 190 mm. Hg. His pulse did not alter in character during his suffering, and the height of the waves showed that there was no contraction of the artery, nor did it differ in character from the pulse tracing taken when he was free from pain. I gave him amyl nitrite to inhale, the pulse quickened (Fig. 169), and the amyl nitrite gave him instant relief. Fifteen minutes after he was quite free from pain, his blood-pressure had risen to 200 mm. Hg., and the

alternating character of the rhythm became more marked (Fig. 170). In the post-mortem account of the patient it will be shown that there was extensive fibrosis of the heart muscle, and disease of the coronary artery.

CASE 2.—Female, aged 60; complained of pain in her chest, radiating into the left arm, and persisting in the left fore-arm with great severity. It was easily induced by exertion, and one day when coming to see me she had hurried. While I was examining her the pain seized her with great violence. I took tracings of her radial and jugular pulses. The heart's rate

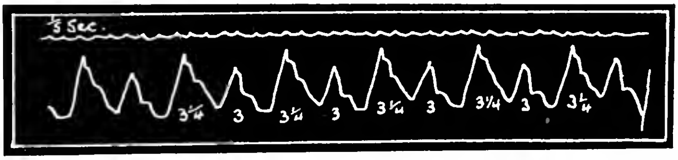


FIG. 170. Fifteen minutes after the inhalation of amyl nitrite and after cessation of the anginal symptoms the alternating character of the pulse became more marked, and the blood-pressure of the larger beats had risen to 200 mm. Hg. (Case 1.)

was increased but the radial pulse was of a good size (Fig. 171). I gave her amyl nitrite and it relieved the pain at once. The patient died three months after from heart failure, and Dr. R. T. Williamson examined the heart for me and found marked atheroma and calcification of the coronary arteries and extensive fibrous changes in the muscle of the left ventricle.

CASE 3.—Male, 52. Mechanical engineer; was going to his work on March 14, 1903, when he was seized with a severe pain across his chest and

FIG. 171. Taken during an attack of angina pectoris—the radial pulse is seen to be of good size. (Case 2.)

called on me. I found him suffering great agony; his face ashen in colour, his pulse full and regular (Fig. 172). I gave him first amyl nitrite with no effect on the pain, then a hypodermic injection of ½ grain of morphia. This gave him relief. The heart's dullness was normal, but the sounds were soft and there was a murmur after the first sound. After a week's rest the patient improved and resumed his work, but on January 15, 1905, he was again seized with a severe attack of angina pectoris. For some months afterwards the pain came on with little provocation, and I saw him in one attack

and the pulse was not affected. The pains diminished in severity as the heart dilated and became weaker, dropsy and oedema of the lungs supervened and he died on June 1, 1905. Dr. Keith's report of the heart stated that the coronary arteries showed a slight thickening of the intima, but the muscle coat was hypertrophied; thickening of the mitral cusps but no stenosis; the foramen ovale was slightly patent; the right ventricle was hypertrophied and dilated, while the left was dilated and atrophied with a considerable degree of fatty degeneration and a slight degree of fibrosis.

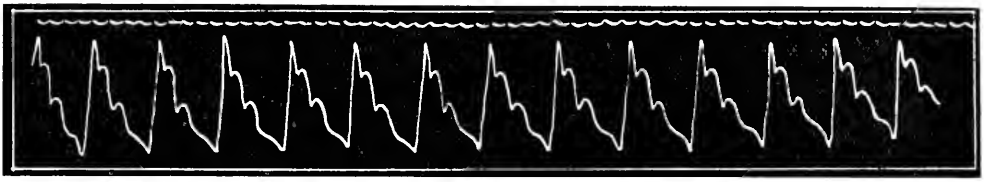


Fig. 172. During an attack of angina pectoris. (Case 3.)

Case 4.—Male, aged 52 at his death; had been under my care for four years. His complaint at first was pain induced by exertion, starting across his chest and radiating into the left arm. At times the pain was so easily excited that he had to keep in bed for a few days. On February 8, 1894, he complained, in addition, of a pain and soreness in the head, which developed into severe attacks of headache. I saw him in an attack of angina pectoris, which came on while he was in bed on March 19, 1894; his pulse was full and regular, and there was no sign of contracted arteries

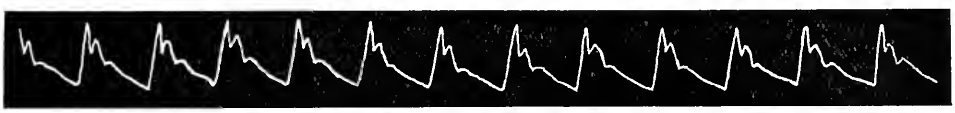


Fig. 173. During an attack of angina pectoris. (Case 4.)

(Fig. 173). I gave him amyl nitrite without effect on the pain, and he was only eased by chloral. He died in an attack of angina on March 27, 1894. Dr. Williamson reported that the ventricles were soft and friable; well-marked atheroma of the coronary arteries; in many places the arteries and their branches were calcified. Microscopic examination of the left ventricle showed fatty degeneration.

Case 5.—Male, aged 68 at his death. Consulted me on November 17, 1899, because of pain over his chest on exertion. He was a millwright by trade—a big powerful fellow. As he was in comfortable circumstances I advised him to give up hard manual work. He followed this advice, and

was free from pain until July 8, 1902, when he was seized with violent pains on going up a hill. He came home and went to bed, when the pain returned, and I saw him during an attack of agonizing severity which lasted over ten minutes. While amyl nitrite was being fetched, I took tracings of his pulse (Figs. 174 and 175). The amyl nitrite gave him a little ease, and the pain gradually subsided. During the attack the character of the pulse did not alter, and there was no sign of contracted arteries. The heart was

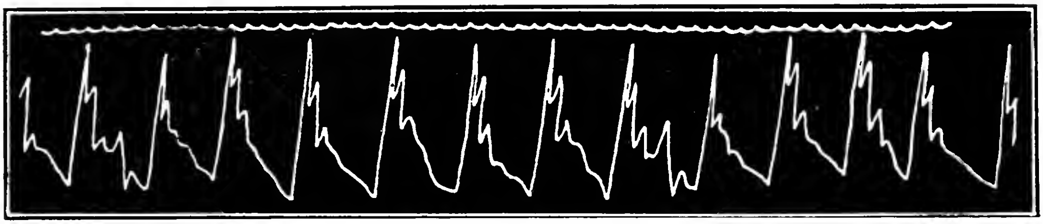


Fig. 174. Large irregular pulse during an attack of angina pectoris. (Case 5.)

irregular, due mainly to extra-systoles, sometimes interpolated as in Figs. 174 and 175. He had repeated attacks and died in one on July 10, 1902.

I have found in a good number of cases that the rate became more frequent during attacks of angina pectoris. In one case of aortic disease the attack of angina coincided with a sudden increase in the rate, of which I got a tracing, but unfortunately it is lost. There is often a tendency to the production of extra-systole induced at the same time, as shown in the

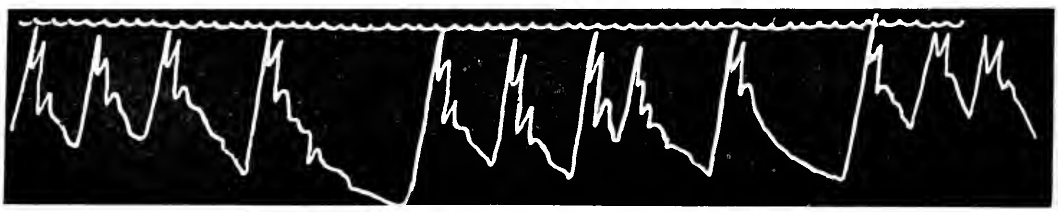


Fig. 175. The same as Fig. 174. (Case 5.)

two last tracings (Figs. 174 and 175). The following case illustrates this tendency :—

Case 6.—Male, 43 years of age : consulted me on September 13, 1900. Had good health until a year ago, when he began to be short of breath on exertion. Shortly after this he suffered from a pain that shot into his left arm when he exerted himself. Four days before coming to me the pain struck with great severity into his chest and down the inside of the left arm, and lasted for half an hour. He has been a heavy drinker. On the 19th

he called again upon me ; was seized with the pain, which held him for some minutes, and I got tracings of the jugular and radial pulses while the pain was present. The pulse increased in rate and became irregular (Figs. 176 and

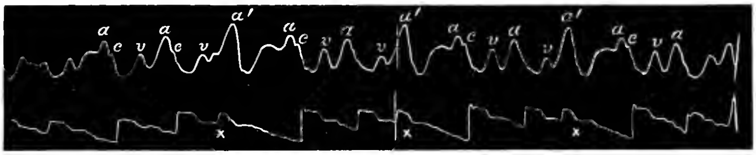


Fig. 176. Shows extra-systoles occurring during an attack of angina pectoris. (Case 6.)

177). These irregularities are due to ventricular extra-systoles ; in Fig. 177 the extra-systole is interpolated between two normal beats.

The patient improved under treatment, and I did not see him again after

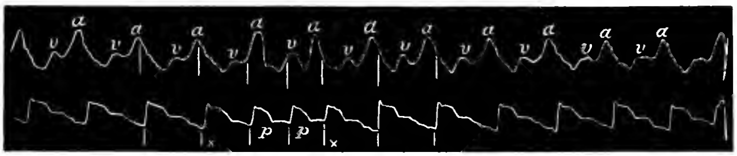


Fig. 177. Shows an interpolated extra-systole during an attack of angina pectoris. (Case 6.)

the end of September. He dropped dead in January, 1901, while watching a football match.

Irregularities of a more obscure kind may appear during an attack of angina pectoris, and the pulse may become slow.

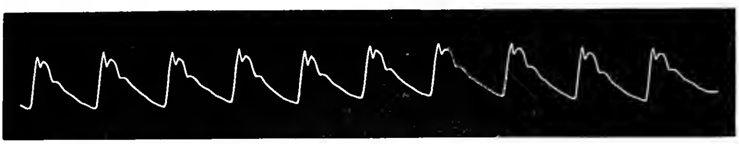


Fig. 178. Pulse of high arterial pressure from a case of chronic albuminuria. (Case 7.)

Case 7.—Female, aged 68. Had been under my care for five years, suffering from cirrhotic kidney. Her pulse was always hard and regular (Fig. 178). On February 28, 1900, she was seized with an attack of angina

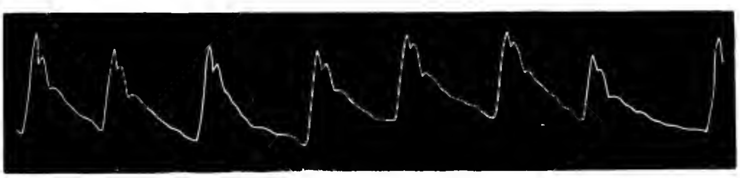


Fig. 179. Irregular pulse during an attack of angina pectoris. (Case 7.)

pectoris. These attacks kept recurring, though she lay in bed. I saw her in an attack on the 25th ; her face was blanched and shrunk, and damp with perspiration. The pulse became very irregular, as in Figs. 179 and 180.

Relief was only obtained by large doses of opium, vaso-dilators having no effect (amyl nitrite, whisky and hot water). Next day she was better and

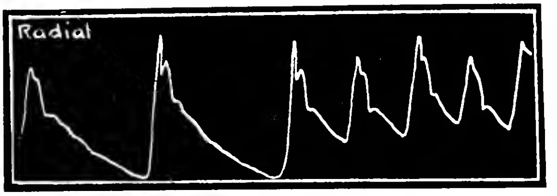


FIG. 180. Tracing of radial pulse showing the appearance of the pulsus alternans after a long pause during an attack of angina pectoris. (Case 7.)

her pulse quite regular (Fig. 181). She died in the following week during an attack of angina pectoris.

The nature of the irregularity in Fig. 180 is easy to understand. An

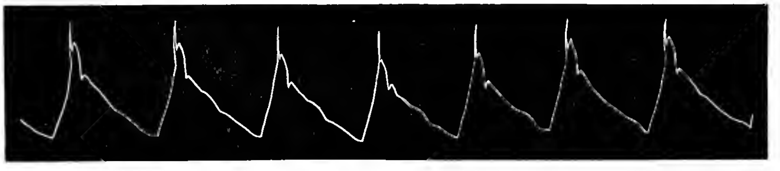


FIG. 181. Pulse of the same patient after the subsidence of an attack of angina pectoris. The pulse is here regular. (Case 7.)

extra-systole occurred during the long pause, and this was followed by the pulsus alternans—an indication of profound exhaustion of contractility. I am not able to account satisfactorily for the nature of the irregularity in Fig. 179.

APPENDIX II

The Nodal Rhythm

The starting of the contraction of the heart at a place other than the normal is a conception so novel in the physiology of the heart's action, and has such a profound bearing upon the diagnosis and treatment of heart failure, that it is necessary to give the evidence for this conception in some detail. Already experimental evidence is accumulating in favour of this idea, as shown by the observations of Cushny[187] (Figs. 58 and 59) and Lewis[207]. Further, the most accurate of all methods of clinical observation, viz. by means of the electro-cardiogram, has amply confirmed the conclusions I have drawn from the study of the heart murmur and graphic records of the circulatory movements (see Appendix VII). Clinical and pathological evidences are given in this Appendix, the former full and complete, the latter confirmatory, and awaiting further work.

The clinical evidence consists in the disappearance of all signs of the auricular systole occurring at the normal period in the cardiac cycle. Such signs of normal auricular activity are :—

(1) A wave in the jugular pulse tracing due to a contraction of the right auricle.

(2) A wave in the apex tracing due to a contraction of the left auricle.

(3) A wave in the liver pulse due to a contraction of the right auricle.

(4) A presystolic mitral murmur due to a contraction of the left auricle.

(5) A presystolic tricuspid murmur due to a contraction of the right auricle.

The most important evidence is found in the character of the jugular pulse. As has been already pointed out, there is normally a wave preceding the carotid and radial pulse, as in Fig. 183, where the wave, *a*, from its position in the cardiac cycle, is recognized as being due to the auricular systole. When the nodal rhythm arises the character of the venous pulse at once changes, and we get a form of venous pulse in which there is no evidence of an auricular contraction in the normal period ; in other words, the jugular pulse changes from one of the auricular type to one of the ventricular type (§ 115), and the heart's action becomes irregular. Presystolic

murmurs due to the auricle, as well as auricular waves in liver and apex tracings, disappear.

In the clinical history of the cases cited I will only refer to the evidence bearing upon the matter in hand. These cases are selected to show that every one of the above-mentioned signs of auricular activity disappear.

I have now collected records of nearly 600 cases of nodal rhythm, and a careful analysis of these leads me to conclude that there are a number of distinct varieties. Most of these I am unable yet to classify with sufficient precision, but I entertain no doubt that with extended experience and improved methods this classification will be ultimately attained. One small class stands out from the others very clearly. In the majority of cases of nodal rhythm the rate of the heart-beat is increased, sometimes extremely so. In a few cases it is found slower than normal, sometimes markedly so. These cases of infrequent rate I place in a separate class and describe them under the term Nodal Bradycardia (Appendix IV).

I had hoped to have been able to give a fairly full account of the pathological condition in cases of the nodal rhythm, but I find there is a vast amount of pioneer work still to be done, and the pathological results given here are put forward with all reserve and are suggestive rather than conclusive. Monckeberg[312] has recently published a book on the pathology of the auriculo-ventricular bundle, but the clinical records are so scanty that no clear perception can be obtained of the symptoms during life, but, so far as I can see, his observations seem to confirm those of Keith given here. For some years I have sent to Professor Keith hearts affected with many forms of disease. He has examined these without knowing the clinical histories. On comparing his descriptions with the clinical notes, I found, with one exception, that all cases that had the nodal rhythm during life presented some evidence of change in the primitive cardiac tissue, or in the artery supplying it. There were also found such changes in the auricular wall in a few cases as to suggest an interruption of the means of communication between the sino-auricular and auriculo-ventricular nodes (Fig. 2). The exceptional case I have subsequently found to belong to a different category (nodal bradycardia, Case 22).

I would particularly draw attention to Cases 11 and 12, which represent typical instances of paroxysmal tachycardia, the one secondary to rheumatic sclerosis, and the other associated with arterial degeneration. The pathological conditions found in these cases afford support for the view put forth that the a.-v. node or bundle, by the invasion of the diseased processes, is rendered more irritable than the sinus remains, and on account of its excessive irritability starts the rhythm of the heart.

Before entering into a detailed account of the cases, there are two points to which it may be useful to refer for the help of those who may follow this line of inquiry. In many cases it will be found that when the radial pulse-beats are very small or occasionally absent, the jugular or liver pulse may be large, as in Figs. 66, 158, 160, and 209. The reason for this is that the force opposing the contraction of the left ventricle (the aortic pressure) is so great that the ventricular contraction is barely able to overcome it. On the other hand, the force opposing the backward flow through the incompetent tricuspid orifice is so slight (the pressure in the great veins) that the right ventricle has no difficulty in overcoming this pressure even with a feeble contraction.

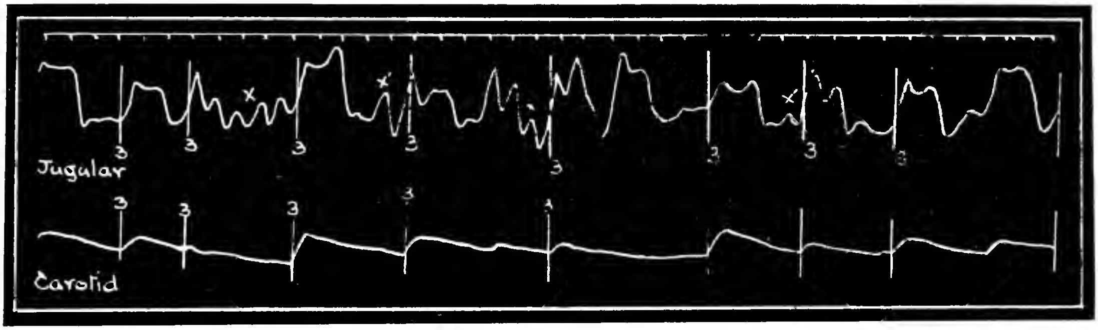


FIG. 182. At × and ×′ a series of waves is shown which were produced by partial compression of the vein by the receiver. The stream of blood passing through the narrowed lumen caused a perceptible thrill in the surrounding structures, and produced vibrations of the tambour which are registered as waves in the tracing. With care the tracing can be obtained free from these artificial waves, as in Fig. 53, which was taken from the same patient.

I have in a few cases of nodal rhythm found in the tracings during ventricular diastole a series of small waves. In the first few cases I sought carefully to see that there was not some error in the method, and failing to find any fault I inferred that these waves were due to fibrillation of the auricle, and I have so described them in one or two published articles. From one patient with a large, full, jugular vein I obtained sometimes very large waves during a long pause, as at × in Fig. 182. When I carefully inspected this vein I found that when I partially compressed the vein with my finger perceptible thrills in the surrounding structures were produced, because the blood was flowing through the constricted vein. From this observation, I now recognize that what I had taken for waves due to fibrillation of the auricle may in some instances have been due to a fault in the method of registration, wherein by compressing the vein with the receiver I had artificially produced thrills which appeared as waves in the tracing.

Other waves due to the same cause are apt to appear, and if the cause be not recognized it might be assumed that the waves were due to the auricle, as at ×′ in Fig. 182.

CASE 7.—*Sudden inception of the nodal rhythm shown by the disappearance of the auricular wave from the jugular pulse, and of the presystolic mitral murmur, with the appearance of permanent irregularity in the heart's action, and*

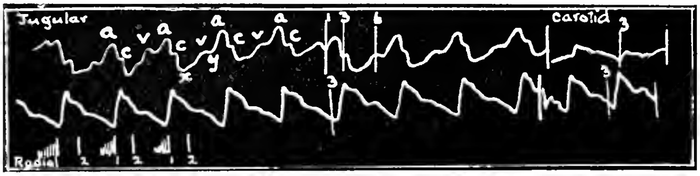


FIG. 183. Simultaneous tracings of the jugular and radial pulses in the first part, of the carotid and radial in the latter part. The jugular tracing shows the form characteristic of the auricular venous pulse where the wave, *a*, due to the auricle precedes the carotid wave, *c* (downstroke 3). The shading underneath represents the time of the presystolic murmur. (Case 7, November 5, 1895).

ventricular form of the venous pulse. Post-mortem evidence of disease affecting the remains of the primitive cardiac tissue.

Female, born 1864. I first saw this patient in 1895, she being then thirty-one years of age. There was a history of acute rheumatism in her youth, and she had marked mitral stenosis. She became pregnant in 1896,

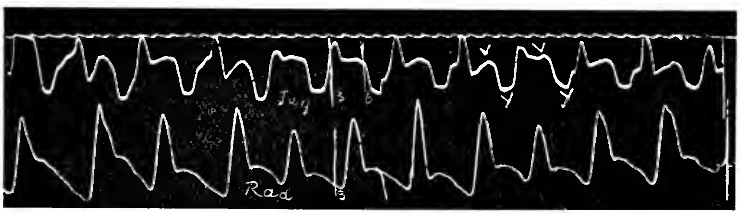


FIG. 184. The jugular pulse is now of the ventricular form—no auricular wave precedes the downstroke 3, and the rhythm is irregular. (Case 7, March 19, 1904.)

and I watched her during the pregnancy and puerperium. She had a long illness from gastric ulcer in 1899. During these years I made frequent observations on her heart. It was invariably regular, and the jugular pulse always showed a well-marked wave due to the systole of the right auricle. There was at first a presystolic mitral murmur of the crescendo type, and latterly a long murmur following the second sound and running up to the crescendo presystolic murmur. The position of the presystolic murmur in the cardiac cycle is diagrammatically represented in the shading under the radial tracing in Fig. 183. This perfect regularity of the heart's action

continued until she suffered from an attack of heart failure in 1900. Coincident with this failure the heart's action became irregular, and the jugular pulse showed no sign of a wave due to the auricular systole at the normal period of the cardiac cycle (Fig. 184). The crescendo presystolic

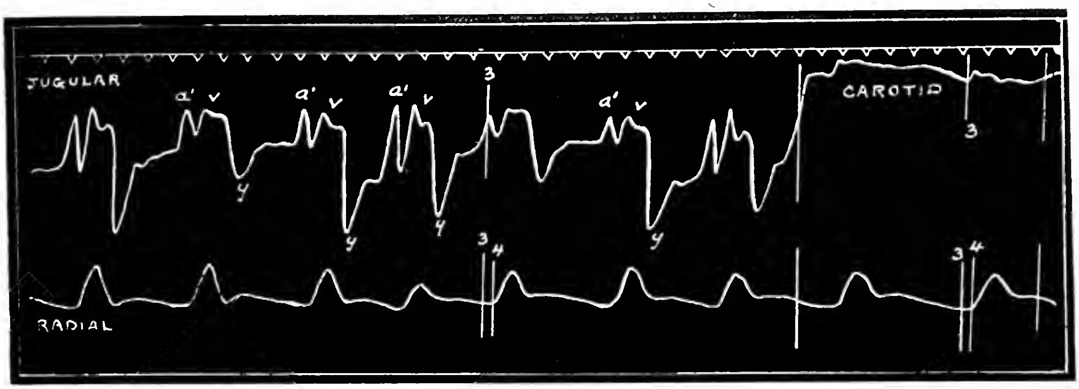


FIG. 185. The jugular pulse is of the ventricular form, and a' is probably due to the auricular systole occurring at the same time as the ventricular. (Case 7, December, 1906.)

murmur had disappeared, while the diastolic portion persisted, as shown by the shading under the apex tracing in Fig. 186.

From this date until she died in February, 1907, these altered conditions persisted, Fig. 185 being a simultaneous tracing of the jugular pulse and

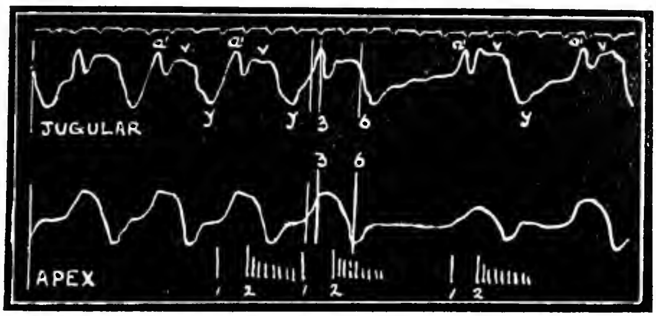


FIG. 186. The jugular pulse shows the same features as in Fig. 185. (Case 7, February, 1907.)

radial taken in December, 1906, and Fig. 186 of the jugular and apex beat taken February, 1907, shortly before she died.

Concerning the jugular pulse, it will be seen that in Figs. 184, 185, and 186, there is not the slightest sign of the auricular wave, a, at its normal period in the cardiac cycle, which is so marked a feature in Fig. 183. The

rhythm of the heart was sometimes fairly regular, but invariably after a few beats the diastolic period showed distinct variations in length; among a large number of tracings which have been taken from this patient since 1900, I have never found one that was regular.

The following is an abbreviated report on the post-mortem examination of the heart:

Valves: Great stenosis of the mitral valves; tricuspid valves shallow and incompetent; pulmonary and aortic valves normal. Coronary arteries healthy; coronary veins dilated to twice their normal diameter. Superior and inferior vena cavae much dilated. The sino-auricular node is normal, but the auricular wall below it is atrophied and fibrous. The taenia terminalis is hypertrophied. The auriculo-ventricular bundle has partly assumed the characters of the ordinary muscular fibres, and has been stretched, and the a.-v. node is flattened by the great inter-auricular pressure. The central fibrous body is marked (fibrous and contracted), and the artery perforating it is atheromatous. The apical half or two-thirds of the left ventricle shows extensive fibrosis. The fibrotic process approaches and incorporates the musculature along a sharply defined line. In the fibrotic tissue are nodules characteristic of rheumatic conditions. Since the fibrotic process is most marked at the base of the musculi papillares, it is possible that it may have spread from the mitral valves.

CASE 8.—*Sudden inception of the nodal rhythm, shown by the disappearance of the auricular wave from the liver pulse, and of the presystolic mitral murmur, with the appearance of permanent irregularity in the heart's action and ventricular form of the liver pulse. Post-mortem evidence of the invasion of the primitive cardiac tissue by the diseased process.*

Female, born 1851, came under my care in 1880. She had had rheumatic fever in childhood, and had suffered from mitral stenosis, there being a well-marked presystolic murmur. In 1880 and 1882 I attended her for attacks of rheumatic fever. In 1892 there was considerable enlargement of the liver, which pulsated (Fig. 187).

The character of the pulsation being of the auricular type, I concluded that there was also tricuspid stenosis, a diagnosis which was afterwards verified at the post-mortem examination. Occasionally I detected an extra-systole, Fig. 189, which Fig. 190 shows to be due to premature contraction of the ventricle. This characteristic pulsation of the liver continued until 1898 (Fig. 188, for instance, being taken in 1897), when she had an attack of heart failure, during which time her pulse was rapid and irregular (Fig. 191).

When the heart had quieted down the tracing showed a complete absence of the wave due to the auricular systole (Fig. 192), and the presystolic murmur had disappeared.

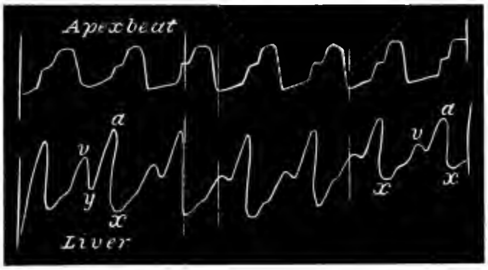


FIG. 187. The liver pulse shows a well-marked wave (a) due to the auricle. (Case 8. 1892.)

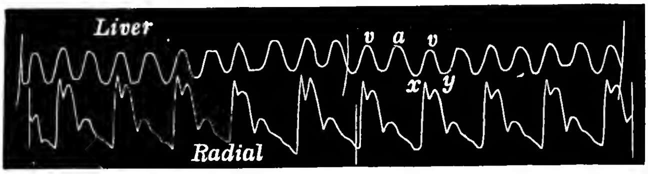


FIG. 188. There is still a well-marked wave in the auricle. (Case 8. 1897.)

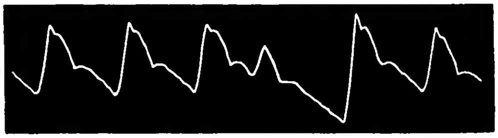


FIG. 189. Shows an extra-systole. (Case 8.)

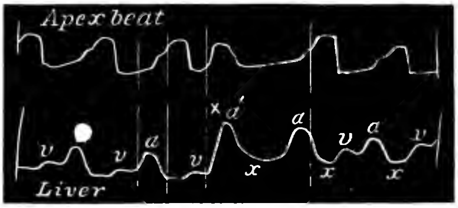


FIG. 190. Simultaneous tracings of the apex beat and of the liver pulse, showing the rhythmical contraction of the auricle, a', while there is a premature beat in the apex (ventricular extra-systole). (Case 8.)

This patient lived until 1899, and in the numerous tracings taken up to her death the rhythm was never again found regular, nor was there present either the presystolic murmur or the wave in the liver pulse due to

the auricle. Here, again, the irregularity in the action of the heart was coincident with the disappearance of all signs of auricular systole, namely, the disappearance of the auricular wave from the liver pulse, showing the cessation of the action of the right auricle, and the disappearance of the presystolic murmur, showing the cessation of the action of the left auricle.

The report of the post-mortem examination of the heart is as follows :—Extreme stenosis of mitral and tricuspid orifices ; great dilatation of auricles,

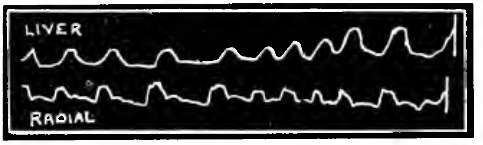


FIG. 191. Sudden inception of the nodal rhythm, showing increased frequency of the heart's action, irregularity and disappearance of the auricular wave from the liver pulse. (Case 8. 1898.)

with atrophy of the musculature. There is a large nodular mass of endocardial thickening showing calcareous masses in its centre, and active inflammatory proliferation at its periphery, on the endocardium, under the aortic orifice, and situated in the pars membranacei septi right over the a.-v bundle. At one point the inflammatory extension has invaded and involved a great part of the bundle. The stretching of the bundle is extreme, and

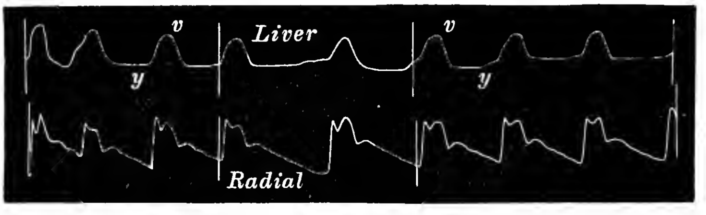


FIG. 192. When the heart became slower the nodal rhythm persisted. When compared with Figs. 187 and 188, it will be seen that there is no auricular wave in the liver pulse, and the heart's action is irregular. (Case 8. 1898.)

there is cell proliferation which Professor Keith regards as due to changes in the walls of the small vessels.

There is extreme atrophy of the upper part of the inter-ventricular septum. It measures only 4 mm. instead of 14–18. The sub-endocardial tissue is thickened, and in parts the Purkinje system is fibrosed and atrophied.

CASE 9.—*Disappearance of large auricular waves from the venous pulse with the onset of continued irregular action of the heart. Fibrosis of the a.-v. bundle.*

Female, born 1850 ; enjoyed good health till 1900, when she began to be short of breath. I saw her first in November, 1902. She was then

very weak, and had to lie in bed propped up ; the legs and abdomen were swollen and the urine scanty ; the pulse was small, weak, and regular, and there was a large pulsation in the veins of the neck (Fig. 193). The heart's dullness extended two inches to the right of the mid-sternal line, and one inch to the left of the nipple line. The sounds were clear and free from murmur.

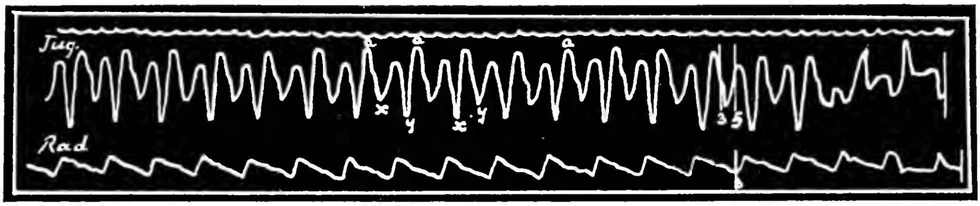


Fig. 193. Shows a well-marked auricular wave in the jugular tracing during an attack of extreme heart failure. (Case 9. 1902.)

Under treatment she improved very much, the pulse becoming slower (Fig. 194), but she had several relapses, until the final breakdown in November, 1904. Figs. 193 and 194 are tracings of the radial pulse and jugular pulse taken in 1902. The jugular pulse is of the auricular form, and the rhythm there is perfectly regular. I took a large number of tracings at different times up till November, 1904, and the rhythm was invariably regular,

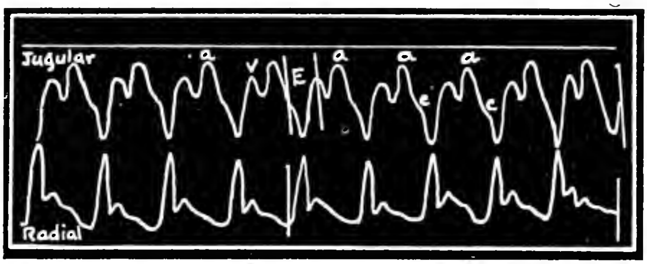


Fig. 194. Shows a well-marked auricular wave (a) in the jugular tracing. (Case 9. 1902).

and the jugular pulse of the auricular type. On one occasion after digitalis she developed a pulsus alternans (Figs. 164 and 165). The breakdown at the last-mentioned date was the most severe she ever had, the legs and abdomen being enormously swollen and the pleural cavities containing a large quantity of fluid. The pulse was now continuously irregular, and the venous pulse had completely changed its character, being of the ventricular form (Fig. 195). She died in December, 1904, two months after I had detected the presence of the nodal rhythm.

The report on the post-mortem examination of the heart is as follows :—

Greater part of auricle has been left behind in subject.

The right ventricle is of quite average length and atrophied ; left ventricle is of quite average length and dilated and atrophied.

Arteries.—Show patches of thickening and dilatation, but nowhere is lumen so reduced as to greatly impede circulation. Aorta shows patches of atheroma.

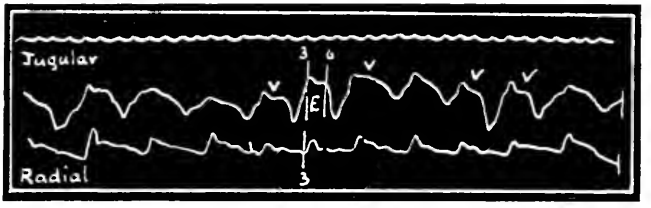


Fig. 195. Simultaneous tracings of the radial and jugular pulses taken on November 9, 1904. The jugular pulse is now of the ventricular type, and the rise in the jugular during the ventricular systole (space *E*) is in marked contrast to the fall during this period in Fig. 194. There is no wave due to the auricle, and the rhythm is continuously irregular (nodal rhythm). (Case 9. 1904.)

Orifices and Valves.—Valves healthy; mitral orifice 29 mm. diam., tricuspid 30 mm., both dilated.

Musculature.—There is atrophy and perivascular fibrosis ; this is extensive in basal part of left ventricle, especially at upper border of interventricular septum, but elsewhere fibrotic changes not marked.

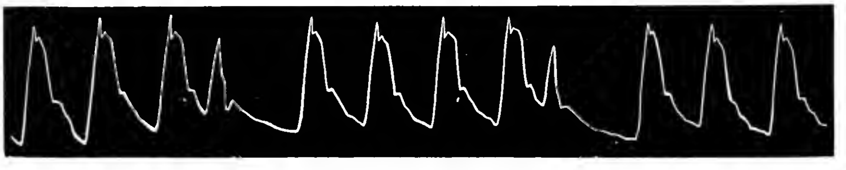


Fig. 196. Radial tracing showing small beats due to the premature contraction of the ventricle (extra-systoles). (Case 10. 1892.)

A.-v. system.—The network at beginning of bundle is normal in size and form, although certain cells which seem inflammatory in nature are present. The bundle, on the other hand, is stretched and small, the fibres show no reticular structure, and in parts show fibrosis. The Purkinje system is overlaid by a very thick fibrous endocardium. There is a marked degree of stretching of the apical half of the left ventricle, and the trabeculae are thin and atrophied ; on section, some of the Purkinje fibres are seen to be undergoing fibrous changes.

CASE 10.—*Frequent extra-systoles from* 1892 *till* 1906. *Signs of contraction of the left auricle in the apex tracings and of the right auricle in the jugular. Sudden disappearance of these signs of auricular activity on the inception of the nodal rhythm in* 1906.

Female, born 1840; came under my care on May 1, 1892, complaining of weakness, shortness of breath, and depression. The radial pulse was

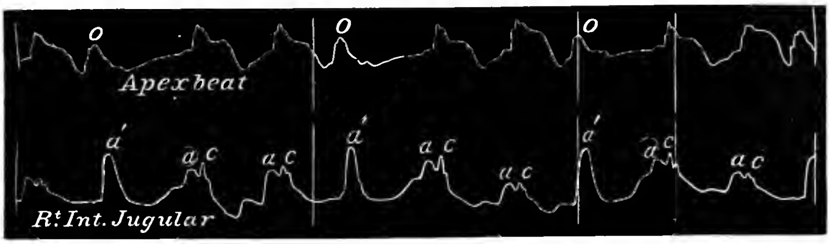


FIG. 197. Shows premature contractions of the ventricle, *o* (extra-systoles), in the apex tracing, while in the jugular tracing are shown waves, *a′*, due to the auricular systole occurring at the same time as the premature ventricular contractions. (Case 10. 1892.)

large and slightly collapsing; there was capillary pulsation on rubbing the forehead, and pulsation in the jugular vein. The heart rhythm was frequently interrupted by extra-systoles, Fig. 196, and during the occurrence of an extra-systole a big wave appeared in the vein. Simultaneous tracings of the jugular pulse and of the apex showed these extra-systoles to be due to the premature contraction of the ventricle. (Fig. 197).

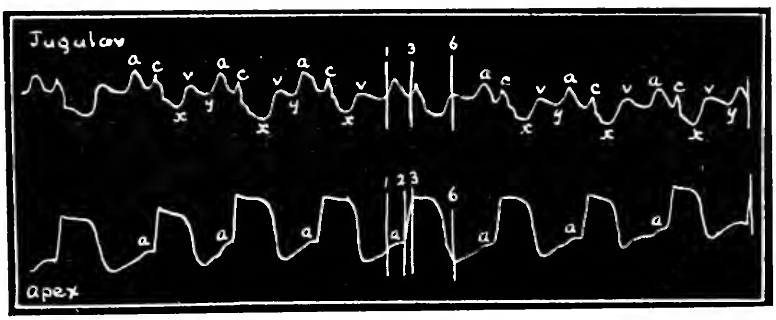


FIG. 198. Shows well-marked auricular waves (*a*) in the jugular and apex tracings. (Case 10).

The heart was only slightly enlarged, and there was a faint systolic and a well-marked diastolic murmur in the aortic area.

This patient's condition varied very much during the later years of her life. She suffered greatly at times from severe attacks of angina pectoris, but no change could be detected in the rhythm of the heart. At other periods the extra-systoles, like those in Figs. 196 and 197, were

very frequent. The jugular pulse was invariably of the auricular type as shown in Fig. 198. In the apex tracings Figs. 197 and 198 there are shown well-marked auricular waves (a), due to contraction of the left auricle.

In July of 1906 she was seized with breathlessness and great prostration. I found the heart extremely rapid and irregular in its action (Figs. 199, 205, Plate II, and 206, Plate III). She regained a certain amount of strength when the heart slowed down, which it did after a couple of months, and Fig. 200 is a tracing of the apex and jugular taken in November, 1906, and Fig. 208, Plate III, is a tracing of the radial and jugular taken at the same time. The heart never dilated very much, and there was never any dropsy, but she died from exhaustion in February, 1907. Figs. 198 and 200 are simultaneous tracings of apex beat and jugular pulse, and the contrast in

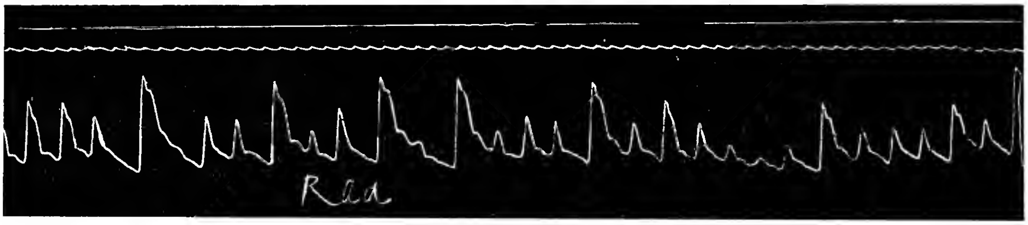


Fig. 199. Rapid irregular pulse due to the sudden inception of the nodal rhythm. (Case 10, July, 1906.)

the two tracings is very marked. In Fig. 198 the rhythm is regular and there is a wave, due to the left auricle, in the apex tracing, and a wave, due to the right auricle, in the jugular tracing. In Fig. 200 there is no sign of the movement of auricle either in apex or jugular tracing and the rhythm is irregular. Figs. 199, 205 Plate II, and 206 Plate III are characteristic examples of the heart rhythm and rate on the sudden inception of the nodal rhythm in many cases.

Case 11.—*Transient attacks of nodal rhythm (paroxysmal tachycardia), slight at first, but becoming permanent and causing death. Post-mortem examination showed involvement of the a.-v. bundle in the disease process.*

Male, born 1860. This patient first consulted me in January, 1900. He had an attack of rheumatic fever at fourteen years of age. In 1896, after walking for twenty minutes at a rapid pace, he felt extremely weak and exhausted. After this he was always short of breath on exertion. A year later, immediately after throwing a cricket ball, he felt the heart flutter for a few seconds. Ten minutes after this the heart ‘ fluttered and beat quickly for six or seven hours ’. He has had an attack of this kind every two or

three weeks since. These attacks last from a few minutes to thirty hours. At first he could sometimes stop the attack by bending down and taking a deep breath, but this does not act now. Sometimes he passes a large quantity of clear urine in the course of an attack. During an attack, if in bed, he feels exhausted and limp, if walking he is easily tired, and if he has to work for some hours he feels swollen round the waist and very sore over the upper part of his abdomen, and feels pain sometimes very severe across the back under the shoulder-blades. During the night the sleep is disturbed. The pulse during these attacks has varied under my observation from one hundred and seventy to two hundred and twenty beats per minute. When the heart is acting quietly a short presystolic murmur at the apex and also a soft diastolic murmur over the middle of the sternum can occasionally be detected. It often happens that these murmurs cannot be

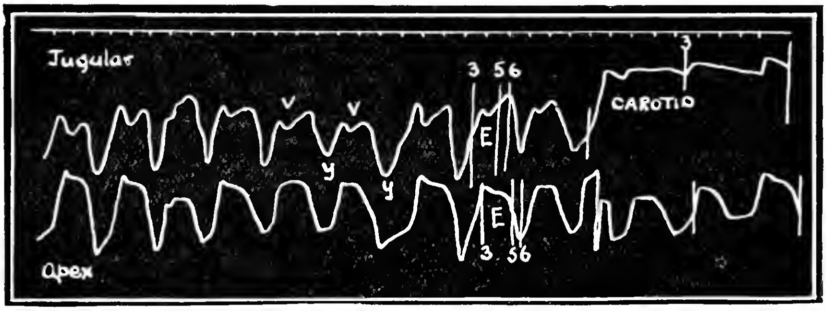


FIG. 200. Here there is no sign of an auricular wave in either jugular or apex tracing. Compare the jugular and apex tracings with Figs. 197 and 198. (Case 10, November, 1906.)

perceived. On producing slight redness by rubbing the forehead, the capillary pulsation can be readily seen. Occasionally the pulse-rate falls to forty-eight beats per minute. Towards the end of an attack, irregularity of the pulse has been detected, due to exhausted contractility (pulsus alternans) (Fig. 130). The patient (who was a very intelligent man) stated that sometimes when the attack of rapid heart-action ceased the heart gave three or four violent beats at intervals longer than the usual pulse-rate. The patient died on November 21, 1900. During the last four months the pulse-rate continued rapid for days together, during which time he would lie prostrate and exhausted. Sleep could only be got by large doses of morphia. During the last two weeks of his life the heart acted slowly only at rare and brief intervals. Signs of heart failure quickly supervened—the face swollen and livid, and general oedema.

It is only necessary here to call attention to the pulsation in the liver and in the veins. I have made many observations on this patient during numerous

attacks of paroxysmal tachycardia. When he was free from the attack only the faintest movement could be detected in the jugular bulb, and, as Fig. 201 shows, there is only a slight wave preceding the carotid pulse. I was fortunate enough one day to see him within five minutes after an attack began. The pulse at first was not excessively rapid, and the carotid pulsation was fairly evident, and I took a tracing (Fig. 202) of the carotid immediately above the clavicle, from the situation from which the jugular pulse in Figs. 201 and 204 was afterwards obtained. In Fig. 202 it will be noted that no sign

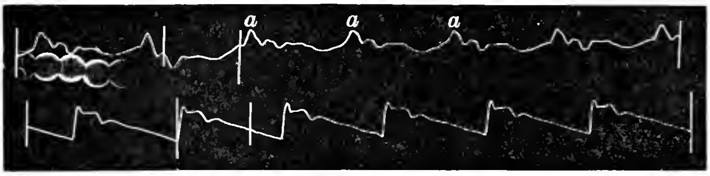


Fig. 201. Tracings of a slight movement in the jugular vein taken at the same time as the radial pulse. The wave *a* is due to the systole of the right auricle. (Case 11.)

of the jugular pulse is present. This would imply that at first the increased action of the heart diminishes the venous pressure, as usually happens when the heart is quickened from other causes. The radial tracing is not so good as I could have wished; still it shows its time relation to the carotid pulse. When I saw him again eighteen hours later the condition of affairs had greatly altered. He had slept little all night; he felt wretched, and

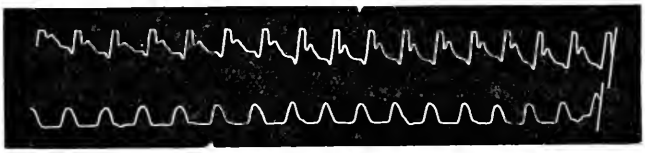


Fig. 202. Simultaneous tracings of the carotid (upper tracing) and radial pulses at the beginning of an attack of paroxysmal tachycardia. The carotid pulse was obtained immediately above the clavicle, from the same place that the jugular pulses in Figs. 201, 203, and 204 were obtained. (Case 11.)

complained of great pain in the upper part of the abdomen and behind about the level of the eighth dorsal vertebra. His face had a wan and weary look, and was of a greyish colour. Above the inner end of the clavicles on both sides there was now visible a large and distinct pulsation. If one carefully palpated this part a distinct pulsating tumour could be felt. It did not extend up the neck, and was quite distinct from the carotid artery. It was evidently the jugular bulb distended by regurgitating waves of blood, and the valves in the jugular and subclavian veins were evidently competent. On auscultating either of these pulsating tumours or under the middle of the

clavicle, one very loud sound was heard, synchronous with each pulse beat. These sounds were evidently originated by the sudden and forcible stretching of the jugular and subclavian valves. When I applied the receiver connected with the tambour over this movement, the lever was jerked up with great violence, and the tracing got had a totally different character from that taken at the beginning of the attack. After taking a few beats from the pulsating jugular bulb, I applied the receiver over the carotid in the middle of the neck, and got the tracing of the carotid pulse (Fig. 203). The wave

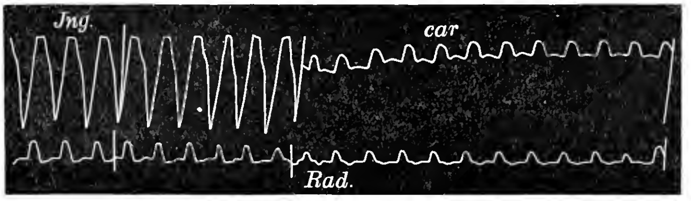


Fig. 203. Simultaneous tracings of the pulsation in the jugular bulb and in the radial, and of the carotid and radial pulses, during an attack of paroxysmal tachycardia, taken eighteen hours from the beginning of the attack. (Case 11.)

obtained from the jugular bulb occurs at the same time as the radial pulse, and must therefore be due to the ventricular systole. I have taken a large number of tracings during many attacks, and they have all presented a similar character. The tracing Fig. 204 was taken after an attack had lasted twenty-four hours. It will be noted that the wave here occurs at the same

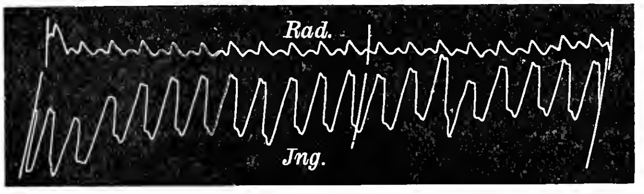


Fig. 204. Simultaneous tracings of the radial and jugular pulses twenty-four hours from the beginning of an attack of paroxysmal tachycardia. (Case 11.)

period as the venous tracing in Fig. 203. That this wave is not due to the carotid can be seen by comparing its character with that of the carotid pulse in Figs. 202 and 203. One could convince oneself that it was not the carotid by examination, the movement being so distinctly limited to the root of the neck, while the carotid above the pulsation could only be found with difficulty. Six months before he died I was able to detect slight enlargement and pulsation of the liver after an attack of tachycardia had lasted fifteen to twenty hours. During the last few weeks of his life he was seldom free from the rapid action of the heart, and the liver extended three inches below

the ribs and pulsated very largely (Fig. 207). As the heart became exhausted the pulse did not acquire so great a rapidity as it did in the earlier stages of the disease, so that we can recognize with certainty the occurrence of the liver pulse during the ventricular systole.

Report of post-mortem examination of the heart :—

Coronary artery healthy ; coronary sinus and veins dilated, but not markedly so.

Left auricle greatly dilated and inter-auricular septum greatly stretched.

Mitral orifice = a linear chink 18 × 3 mm. Anterior cusp of mitral valve and chordae tendineae are the site of a warty hard vegetation, the size of a hazel-nut.

Tricuspid valves healthy, but orifice dilated.

Myocardium = partial fibrosis in areas. Everywhere the small vessels and capillaries are dilated and the nuclei in their walls dividing, and in the

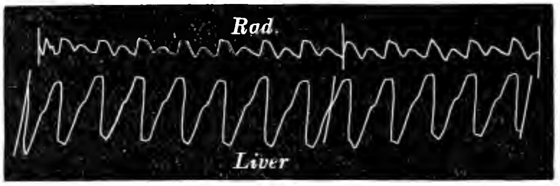


Fig. 207. Simultaneous tracings of the radial and liver pulses during an attack of paroxysmal tachycardia towards the end of life. (Case 11.)

neighbourhood of capillaries are plasma cells. This is markedly the case with the upper part of the a.-v. bundle and lower part of node.

The sino-auricular node is very well marked, but appears in parts to be more fibrous than usual, and the vessels show the proliferation of cells seen elsewhere in the heart.

The pathological process which has affected the mitral valves spread up to the central fibrous body, and where the bundle perforates this there are signs of cellular changes in the margin of the bundle. In some sections of the a.-v. bundle lower down, nearer the ventricles, there are seen in it small areas from which the muscular tissue seems to have disappeared. There are thus distinct evidences of cellular changes in the bundle.

Case 12.—*History of many years of extra-systoles, sometimes becoming very frequent. Transient attacks of the nodal rhythm, slight at first, becoming more prolonged till it became permanent. Died five months after permanent establishment of the nodal rhythm. Post-mortem : obliteration of lumen of artery supplying the a.-v. bundle, and probable disease of the a.-v. bundle.*

Female, born 1846. I have known and attended her for slight ailments at intervals since 1880. In 1892 I obtained tracings of her pulse, which

showed extra-systoles. These occurred sometimes at rare, sometimes at frequent intervals (Figs. 99, 103 and 104, Plate II). They were usually ventricular in origin, but occasionally nodal and auricular. I also noted in 1892 that there was a gallop rhythm of the heart when it was regular. In 1900 she began to have attacks of 'palpitation' of short duration, and I obtained tracings of the radial and jugular during one of these attacks, and they showed a transition of the venous pulse to the ventricular form during the attack. On October 13, 1903, she felt weak and exhausted, and had a distressing fluttering sensation within her chest, and I found the heart's action extremely irregular. The attack lasted four or five hours. The tracings taken during the attack were of the same character as Fig. 209. On October 19 she was again seized with a similar attack, which lasted

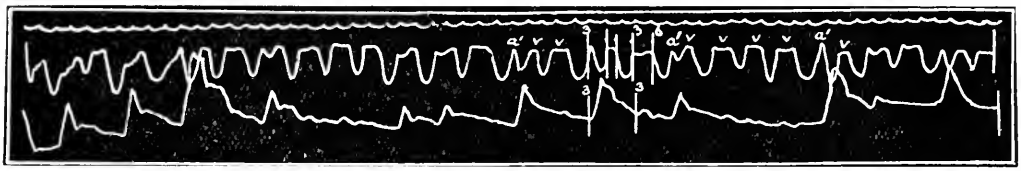


Fig. 209. Characteristic irregularity on the sudden inception of the nodal rhythm—the jugular pulse is of the ventricular form. (Case 12.)

a whole day, and the tracings in Fig. 209 convey a very good idea of the character of the heart's irregularity. The next day the heart was quite regular and the jugular pulse a typical example of the auricular form.

On October 27 the heart again became very irregular. This attack continued without intermission until November 1. On October 29 the heart's action became much slower, but the irregularity still persisted, and the character of the jugular pulse showed a curious change (Figs. 210 and 211), viz., during the ventricular systole (the period between the perpendicular lines 3 and 6) there are two waves a' and v, while there is no wave at the normal time of the auricular wave. The heart suddenly reverted to the normal rhythm, and became regular with a typical auricular venous pulse (Fig. 212).

The attacks gradually lessened in frequency and duration until June 12, 1904, when, after a long walk in the country, she was seized with an attack which lasted for a fortnight. A few days before the attack finally subsided the heart became normal in its rhythm for a few hours. On October 16, 1904, this nodal rhythm again started, and continued, with great dilatation of the heart, dropsy, ascites, and hydrothorax, until her death on March 17, 1905.

In this patient the remarkable changes described in § 159 were seen most typically. A few hours after an attack the heart dilated, the liver enlarged, the face became swollen and livid. Immediately the normal rhythm was restored, the patient at once felt relief, and in a few hours all the abnormal symptoms disappeared.

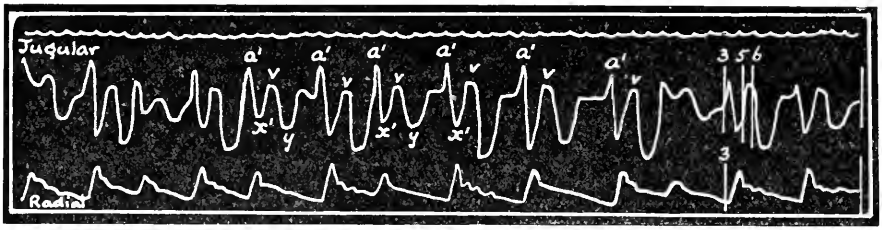


FIG. 210. The rhythm is still irregular and the jugular pulse is of the ventricular type, the auricular wave a' occurring during the ventricular systole. (Case 12.)

As this patient lived close to my house I saw her very frequently, and had the opportunity of watching the attacks begin and finish. Once when taking a tracing the attack started, and on several occasions the attacks ceased while I was watching her. I have accumulated a great number of tracings taken at all stages.

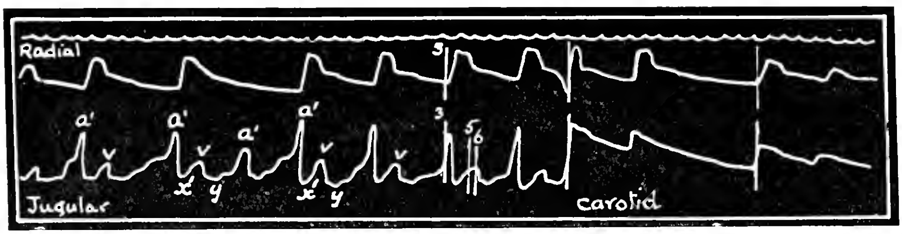


FIG. 211. Shows the same as Fig. 210 except that during the long pause of the ventricle an independent auricular wave (a') appears, not followed by a v wave. (Case 12.)

The character of the jugular tracing showed invariably the typical ventricular venous pulse during the attack, and the return to the auricular jugular pulse with the cessation of the attack. There are a few instructive features in the character of the jugular pulse. The heart's rate varied very much during the attack. At the beginning it was usually very rapid, then it began to get slower in an irregular way ; at first an occasional long diastolic pause would occur, followed by a large ventricular contraction. Then longer pauses sometimes became more frequent, till finally the heart's rate was not much above the normal, though it was irregular in rhythm (Figs. 210 and 211). When the rate was rapid, sometimes there was scarcely any evidence in the radial pulse of the ventricular contraction, while the jugular

pulse showed well-marked waves during the intermission (Fig. 209) in the radial pulse. When the heart was rapid, the wave *v* in the jugular tracing was full during the whole of the ventricular systole; but during slow action of the heart—whether the slow beat was occasional, as in Fig. 209, or continuous, as in Fig. 210—the wave *v* was divided into two portions (*a′* and *v*)

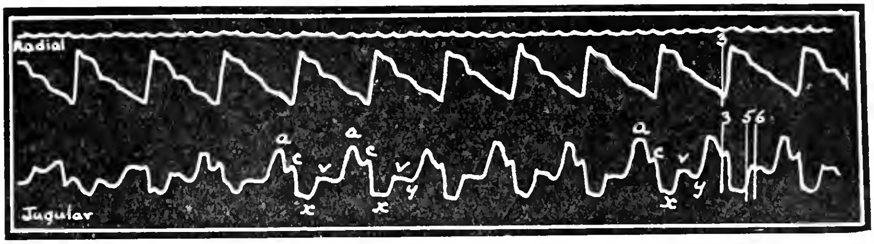


Fig. 212. With the sudden reversion to the normal rhythm the heart becomes regular and the jugular pulse of the normal auricular type. Compare this tracing with the three preceding. (Case 12.)

by a deep depression. The explanation I suggest is that the auricular contraction occurred during the ventricular systole, but, being short, it finished before the ventricular systole (as in Cushny's tracing, Fig. 58), and that the wave *a′* in these tracings is due to the auricular systole, and the fall *x* is due to the auricular diastole (probably the other factors mentioned in § 108 assist

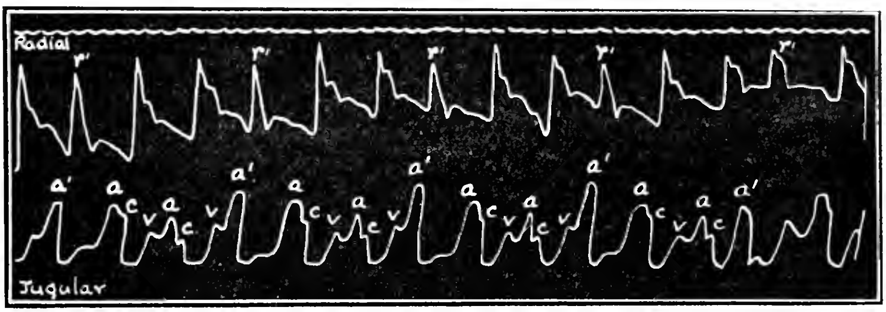


Fig. 213. Sometimes when the heart reverted to the normal rhythm ventricular extra-systoles (*r′*) would appear. (Case 12.)

in deepening this fall). When the heart beats rapidly the engorgement of the right side is so great that it obliterates the effect of the auricular diastole. This explanation of *a′* is supported by the fact that sometimes during a long pause, when the heart was acting slowly and irregularly, a wave, presumably auricular in origin, would appear as in Fig. 211. When the heart suddenly reverted to the normal rhythm, then it was sometimes quite regular, as in Fig. 212, or there was an occasional

extra-systole, sometimes of auricular origin, as in Fig. 99, sometimes ventricular, as in Fig. 213. During the last three months of her life the rate was continuously rapid, and though I pushed such drugs as digitalis and strophanthus no change was produced. Fig. 214 was taken a month before she died.

Report of the post-mortem examination of the heart :—

Arteries.—Left coronary chiefly affected ; lumen narrowed. Anterior interseptal artery which supplies the a.-v. bundle closed completely ; right coronary affected, but to a less degree.

Orifices and Valves.—Valves not diseased. Mitral orifice dilated, tricuspid dilated, inferior caval dilated, aortic normal.

Musculature.—Taenia terminalis hypertrophied. A.-v. node and bundle large ; the fibres have the appearance of being stretched, having lost their stellate reticular form : applied closely, and rather longer than usual. The

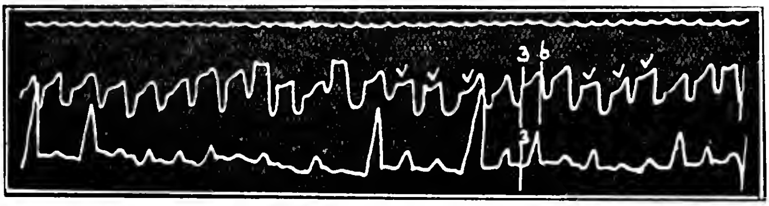


FIG. 214. Shows the character of the persistent nodal rhythm one month before death. (Case 12.)

right and left septal divisions are normal in appearance. The inter-auricular septum is stretched. The apical two-thirds of the left ventricle shows large patches of fibrosis, and the trabeculae with the Purkinje system are stretched, and certainly not healthy—fibrosis and atrophy—result of endarteritis.

CASE 13.—*Frequent attacks of the nodal rhythm, with no serious effects, in old age.*

Male, born 1827. Has suffered from frequent attacks of rapid action of the heart, accompanied by a feeling of great prostration since he was seventy-six years of age. During the attacks he feels very exhausted, and lies in bed ; they last from half an hour to twelve hours. He may be free from them for weeks, at other times he has several in one week. I have seen him in consultation during these attacks and also when quite free from them. In the latter condition he is a hale man, considering his years, and takes an active part in his business ; his heart shows no abnormality, and his pulse is slow and regular (Fig. 215). During the attack he lies very still in bed ; his face is pale and slightly drawn. He does not care

to make much exertion, but has no actual suffering. The pulse sometimes attains a rate of 200 per minute at the beginning of the attack. It was always several hours after an attack had begun before I saw him, and the pulse was usually between 150 and 170 beats per minute. Fig. 215 is a tracing of his jugular and radial pulses while free from the attack; there was very slight pulsation in the neck, and I had some difficulty in getting

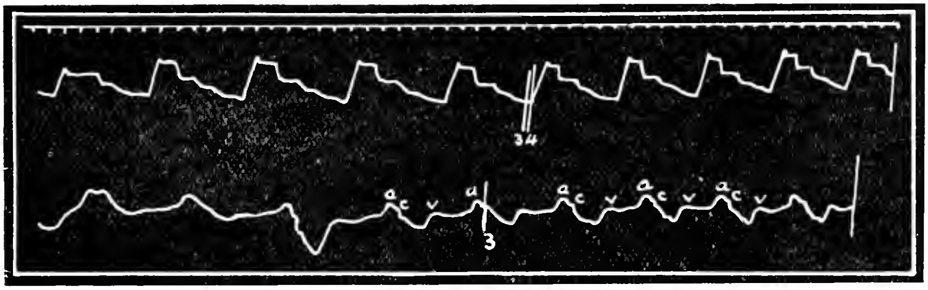


Fig. 215. Tracings from a man aged 78, when free from an attack of paroxysmal tachycardia. (Case 13.)

a tracing. Its character, however, clearly shows it to be of the auricular form. Fig. 216 shows the radial and jugular pulses during an attack. I had considerable difficulty in getting satisfactory tracings, as the patient was in bed in a position not conducive to taking a good tracing, but in all those I took the characters were the same as in Fig. 216. Here the

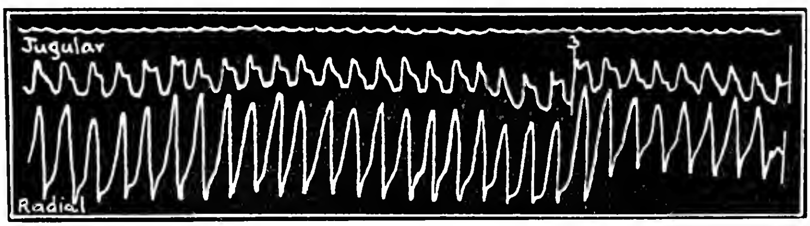


Fig. 216. From the same patient that gave Fig. 215, during an attack of paroxysmal tachycardia. Note slight pulsus alternans in the radial tracing. (Case 13.)

jugular pulse is manifestly of the ventricular type. In the radial pulse there is seen a tendency to the alternans form.

When last I heard of him (1908), at the age of eighty-one, he was in fair health, and still liable to these attacks.

Case 14.—*Attacks of paroxysmal tachycardia.*

Male, aged thirty. Came under my care suffering from a hydatid cyst of the liver. There was no history of any heart trouble, except that for the last two years he had been conscious of a rapid 'fluttering' action of the

heart. I was about to operate on him when I felt his pulse—it was beating at the rate of about 200 beats per minute. He was conscious of this rapid action, and said that it had come on two hours previously when at the closet. The attack ceased quite suddenly two hours afterwards. I lost sight of him some months afterwards, but he seemed in no way impaired by these attacks.

Fig. 217 is a tracing of the jugular pulse when he was at rest. It is a typical instance of the auricular form of the venous pulse. Fig. 218 shows the character of the jugular and radial pulses during the attack. The jugular pulse is now of the ventricular type.

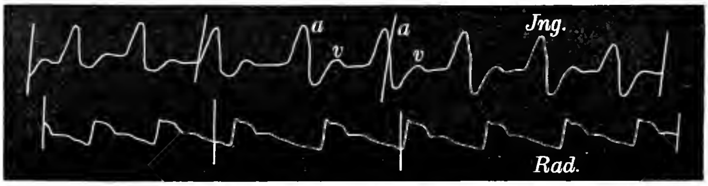


FIG. 217. Simultaneous tracings of the jugular and radial pulses from a case of paroxysmal tachycardia during the quiet period of the heart's action. There is a well-marked auricular wave, *a*, in the jugular pulse. (Case 14.)

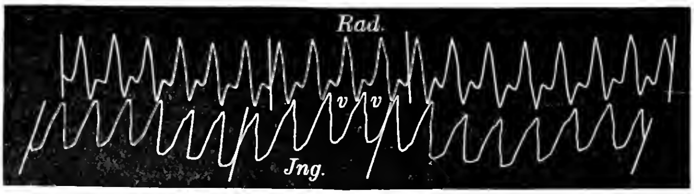


FIG. 218. Simultaneous tracings of the radial and jugular pulses during an attack of paroxysmal tachycardia. The jugular pulse is due to the ventricular systole, there being now no trace of the auricular waves. (From the same patient as Fig. 217. Case 14.)

CASE 15.—*Sudden inception of the nodal rhythm, with persistent rapid heart action, dilatation of the heart, dropsy, death in three weeks.*

Female, aged sixty-five. I had attended her for various ailments (rheumatism, bronchitis, &c.) during a period of over twenty years, and her heart had invariably been quite regular. On June 20, 1904, she returned from the seaside to her home and sent for me. She had been taken ill a few days before, her chief complaint being shortness of breath. When I saw her she was propped up in bed, and breathing rapidly and laboriously. Her pulse was extremely rapid and irregular, but I did not have my polygraph with me. Next day when I called to see her, I found her greatly improved, out of bed, and free from distress. Her pulse was full, regular,

and not rapid (Fig. 219). The following day, however, she was again very bad, and Fig. 220, taken on June 23, gives a good idea of her pulse. She was very stout and short-necked, and I had difficulty in getting a jugular tracing ; her breathing was laboured and, the sterno-mastoid being in action, I could only get an imperfect tracing from the jugular, as in Fig. 220. Imperfect as it is, it shows clearly that the venous pulse is of the ventricular form. I tried all sorts of remedies to slow the heart—digitalis and opium, adrenalin, trinitrin, &c., but all without avail. The heart dilated, dropsy set in and became very extensive, and she died three weeks after the permanent establishment of the nodal rhythm.

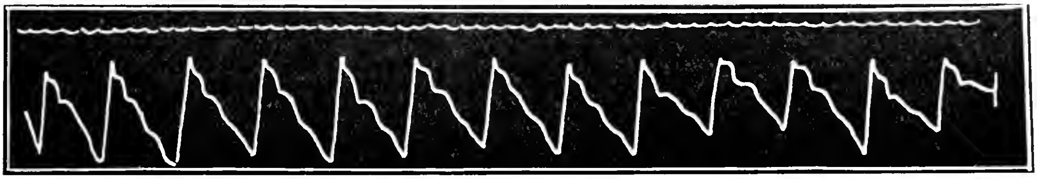


Fig. 219. Regular pulse between attacks of nodal rhythm. (Case 15.)

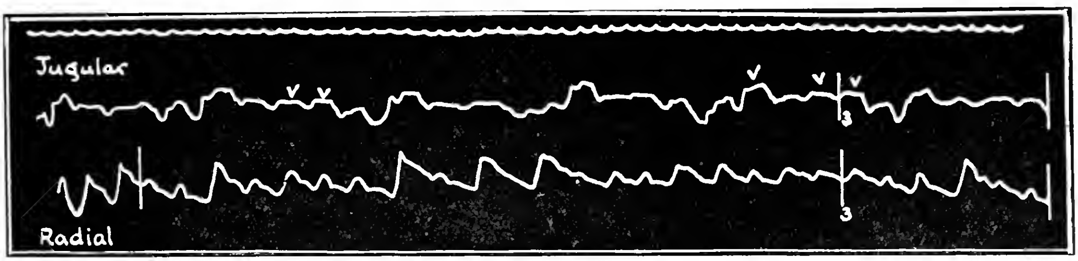


Fig. 220. Permanent inception of nodal rhythm, leading to a fatal issue in three weeks. (Case 15.)

Case 16.—*Frequent attacks of paroxysmal tachycardia without serious symptoms.*

Female, aged thirty-five, eight months pregnant with her second child. For several years she suffered from breathlessness, and was conscious that her heart beat ' very queerly '. She was under my care for only a few weeks, and I saw her during several attacks of paroxysmal tachycardia. After she left me I heard that she had had an easy confinement, and some years later was in fair health, though still at times prostrate on account of her heart. The periods of nodal rhythm were of varying duration, the attacks not being continuous, but interrupted frequently by normal beats. At other times the heart would only show frequent extra-systoles. When the heart was irregular the venous pulse was always large, while when the heart was regular

it was scarcely perceptible, and it was with difficulty that I got the faint tracing of it in Fig. 221. The waves in the jugular, though slight, are recognizable, and the jugular pulse is of the auricular type. In Fig. 222 the heart is acting irregularly. The radial pulse shows three long pauses at × × ×. The auricular waves, *a* and *a′*, in the venous pulse occur at regular intervals. During each long pause in the radial there is a large wave, *a′*, due to the auricle, and larger than the other auricular waves, for the reason already given, p. 153, namely, because at the period at which the auricle contracted

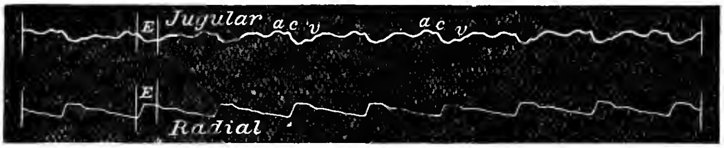


FIG. 221. Simultaneous tracings of the jugular and radial pulses. The jugular pulse is of the auricular form. These and the following four tracings are from the same patient. (Case 16.)

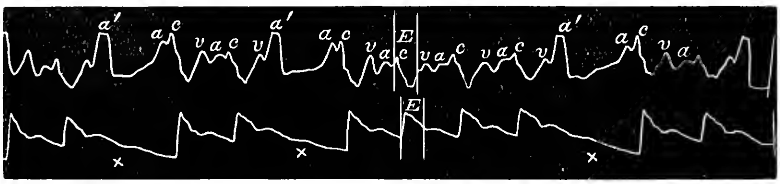


FIG. 222. Simultaneous tracings of the jugular and radial pulses during irregular action of the heart. The auricle preserves its rhythm, there being a large wave, *a′*, during the premature contraction of the ventricles. (Case 16.)

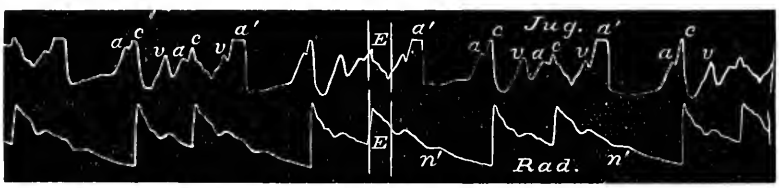


FIG. 223. Shows the same as Fig. 222. (Case 16.)

the ventricle was in systole, and hence a larger wave was sent back into the veins. It will be noted that after the large auricular wave, *a′*, there is never a ventricular wave. This tracing exemplifies the form of irregularity due to premature stimulation of the ventricles alone .(ventricular extra-systole).

In Fig. 223 a very similar irregular condition is present, the difference being that every third arterial beat here is missed, and is represented in the radial tracing by the notch *n′*. In these three tracings (Figs. 221, 222, and 223) the period *E*, representing the time when the semilunar valves

are open, shows in the jugular pulse a great fall. In Fig. 224 the radial tracing shows two normal beats in the centre of the tracing, all the others being nodal in origin. The beat preceding the full beats shows only a notch, *n′*, as in Fig. 222. The venous pulse at the time of the normal radial beats shows the same features as are present with the normal radial beats in the three preceding tracings, namely, a small auricular wave, *a*, the carotid wave *c*, the auricular depression during the period *E*, and the ventricular wave *v*. But when the venous pulse corresponding to the

Fig. 224. Simultaneous tracings of the jugular and radial pulses, showing two norma radial beats in the centre of the tracing. Corresponding to the ventricular systole, *E*, there is a fall in the jugular pulse when the radial beat is normal, and a rise, *E′*, when it is of nodal origin. (Case 16.)

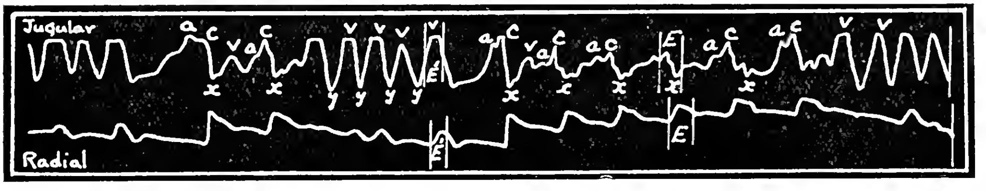


Fig. 225. Shows an alternation of the normal and nodal rhythms. (Case 16.)

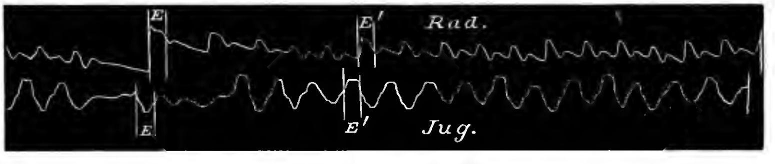


Fig. 226. There is only one normal beat (*E*), all the other beats being of noda origin. (Case 16.)

nodal beats is considered, a remarkable change is found. There is but one large wave and one large fall, and the period when this wave occurs is during the ventricular systole *E′*, in striking contrast to the large depression at the time with the normal radial beats shown in the preceding three tracings. A similar condition is seen in Fig. 225, where there is a continuous variation from the normal rhythm to the nodal. In Fig. 226, with the exception of one normal beat preceded by a long pause, the jugular pulse is of the ventricular form. The transition from one form of jugular pulse to the other is well brought out in Fig. 225.

APPENDIX III

Paroxysmal Tachycardia of Auricular Origin

In reflecting upon the causes of abnormal rhythms of the heart, it seemed reasonable to expect that at the place where an occasional stimulus arose giving rise to an extra-systole it would be probable that the same place might start off the heart's action continuously. This view has been confirmed in the instances of ventricular and nodal extra-systoles being the precursors of the nodal rhythm. I had considered that inasmuch as auricular extra-systoles were of frequent occurrence, it would be but reasonable to expect that the heart's action might in some cases start continuously from the place at which the auricular extra-systoles arose. In preparing the first edition of this book I had examined my records for such a condition and found a number of cases that seemed to fulfil this prediction. In four instances the patients were suddenly seized with great rapidity of the heart's action, and they all died from heart failure after a period varying from one to four weeks. The tracings taken from these patients present considerable difficulty in their interpretation, but I could only conclude that they were cases of auricular tachycardia. In two other cases there seemed distinct evidence of a paroxysmal tachycardia of auricular origin. As the tracings were not altogether convincing I did not allude to the matter, but resolved to wait for further experience. Within the past year cases of undoubted paroxysmal tachycardia have been published by Cowan, Macdonald and Binning [236], and Lewis [251], while a case of continuous auricular tachycardia has been published by Hertz and Goodhart [243], and Dr. W. T. Ritchie has shown me another.

I have at present under observation a very instructive case of this auricular form of paroxysmal tachycardia. The patient, a man aged 41, a carpenter by trade, complains of weakness, palpitation, and attacks of giddiness. The radial pulse is soft and compressible, and shows a varied irregularity. At times there may be an occasional intermission, or these intermissions may be very frequent. At irregular intervals the rate becomes suddenly very rapid, and as suddenly becomes slow. These periods of

rapid action may last for a few seconds or for ten minutes. Tracings taken of the radial and jugular pulses show the intermission to be due, as a rule, to auricular extra-systoles, when the ventricle failed to respond

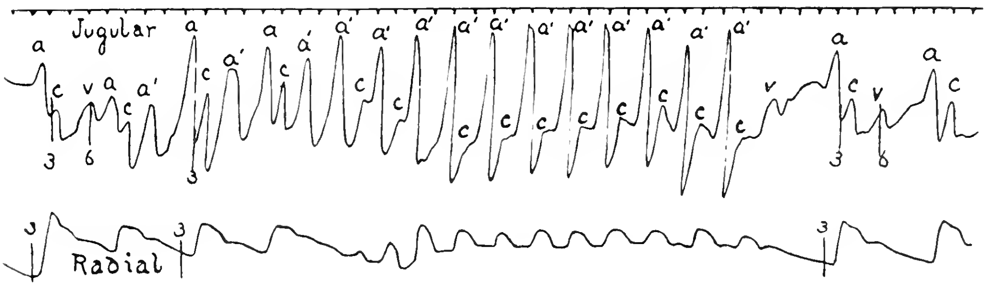


FIG. 227. Simultaneous tracings of the jugular and radial pulses during one attack of paroxysmal tachycardia. The first cardiac cycle in the jugular tracing shows the normal events (*a*, *c*, *v*). The second cycle shows the normal waves *a* and *c*, but the wave following marked *a′* occurs earlier than the wave *v* in the previous cycle, and is due to a premature or auricular extra-systole, but is not followed by a *c* wave or by a radial pulse beat. The next two normal *a*, *c*, waves are each followed by an auricular extra-systole (*a′*) with no ventricular response, as shown by the absence of the *c* wave and the radial pulse beat. These are all ‘ interpolated auricular extra-systoles ’. After these there follows a series of auricular premature beats (*a′*) to which the ventricle responds as shown by the *c* waves and the small radial pulse beats.

The onset of the paroxysm always coincides with great distension of the jugular veins, which is shown in the tracing by the greater amplitude of the auricular waves *a′*.

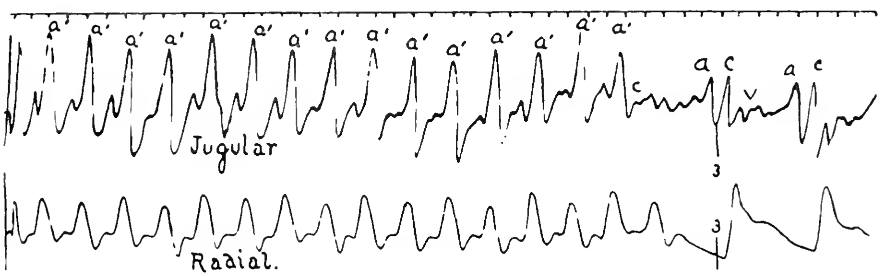


FIG. 228. From the same patient as Fig. 227, taken at the end of a long paroxysm. It shows the same features as Fig. 227, but here there is a well-marked alternation of the radial pulse beats during the paroxysm.

to the auricular extra-stimulus, or responded so weakly that the pulse beat did not reach the radial (Figs. 227 and 228).

The rapid pulse beats in the radial are seen to be in response to a rapid series of auricular contractions—the waves *a′* in Figs. 227 and 228

being due to the abnormal series of auricular contractions. In Fig. 227 one of these short paroxysms is shown, while in Fig. 228 there is shown the end portion of a long attack. The radial pulse shows a well-marked pulsus alternans, a feature not infrequently found after a long paroxysm in the more common form of nodal paroxysmal tachycardia (see Fig. 130, p. 198).

APPENDIX IV

Nodal Bradycardia

I have already remarked that in the vast majority of cases, where the nodal rhythm is present, the heart's action is at first more rapid than normal, but there is a class of case where, so far from the rate of the contraction being more rapid than the normal, it becomes slower, and sometimes very markedly so. In most other respects the character of the heart's action corresponds to the more common forms of nodal rhythm, but if, as seems probable, these commoner forms owe their inception to disease rendering some part of the heart, such as the node, more excitable, this cannot hold good when the rate is much slower than that of the normal rhythmic area. Hence a search has to be made for some other cause. Facts pointing to a definite cause may appear when all the features connected with these cases are studied, and are compared with the results of experiment. There is probably more than one form of this nodal bradycardia, for, in the cases I have collected, in some the pulse though slow is quite regular, while in others it is continuously irregular.

In some of these cases the heart's action becomes so slow that cerebral anaemia results, with all the phenomena characteristic of the Adams-Stokes syndrome, so that they may easily be mistaken for heart-block. The differential diagnosis between cases of nodal rhythm and heart-block lies in this—in heart-block the auricle is active and independent of the ventricular systole, so that there is never a jugular pulse of the ventricular form ; the ventricular form of the venous pulse, on the other hand, is characteristic of the nodal bradycardia.

Case 17.—*Old rheumatic affection of the heart, with long-continued impairment of the a.-v. bundle, with a delay between the As and Vs. Sudden inception of a slow and irregular action of the heart, with disappearance of all evidences of auricular contraction, at first transient, later permanent.*

Male, born in 1851. I attended him for an attack of rheumatic fever in 1883. He was left with a damaged mitral valve, but remained in fair health till 1897, when he had serious heart failure. From this also he made a good recovery, and his heart remained perfectly regular till 1904. I have taken tracings of his apex, radial pulse, and jugular pulse at frequent intervals

since 1892. His heart was invariably regular except for a short period in 1897, the irregularity being due to dropping out of ventricular systoles (mild heart-block, see Fig. 115). His jugular pulse was always of the auricular form, a peculiarity in this case being a persistent increase in the *a–c* interval. Tracings of the jugular and radial pulses, taken in 1892, are given in Fig. 229; in 1903 in Fig. 230. Jugular and apex tracings are

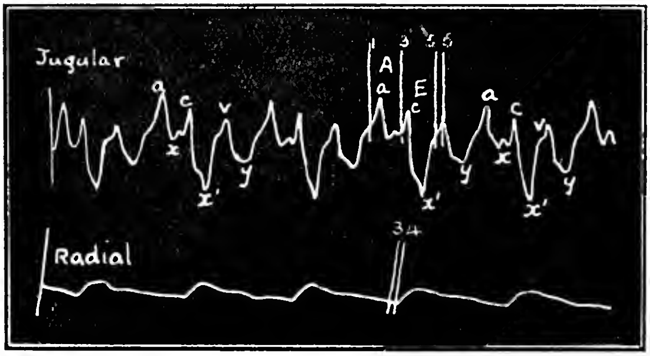


Fig. 229. Shows a regular rhythm and an auricular venous pulse and wide *a–c* interval (space *A*). Taken 1892. (Case 17.)

represented in Fig. 231. The rhythm is regular, and the auricular wave, *a*, is well marked in both apex and jugular tracings. There was a long diminuendo murmur after the second sound, and a well-marked presystolic murmur separated by a brief interval from the first sound (shading in

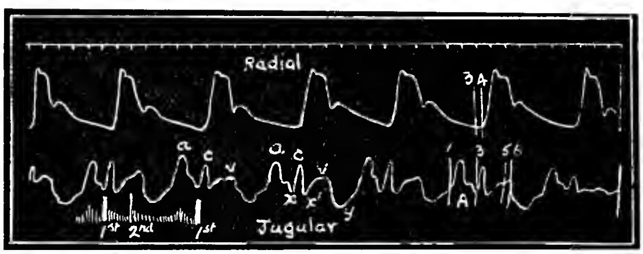


Fig. 230. Shows a regular rhythm and an auricular venous pulse and wide *a–c* interval (space *A*). Taken 1903. (Case 17.) (Figs. 229 and 230 are also discussed in § 164.)

Fig. 230). In the numerous tracings of the apex beat I have taken up to April 19, 1904, there was always a well-marked auricular wave, *a*, preceding the large wave. When he visited me on the last-named date I found his heart continuously irregular, and on taking tracings of the jugular pulse I found it of the ventricular type (Figs. 232 and 233), the presystolic murmur had gone, and there was at the time only a diastolic murmur as shaded in Fig. 232. The wave due to the auricular systole had disappeared from the

apex tracing. Here, again, with the appearance of the ventricular venous pulse and continuous irregularity all evidences of the contraction of the right and left auricles had disappeared. When this patient called to see me

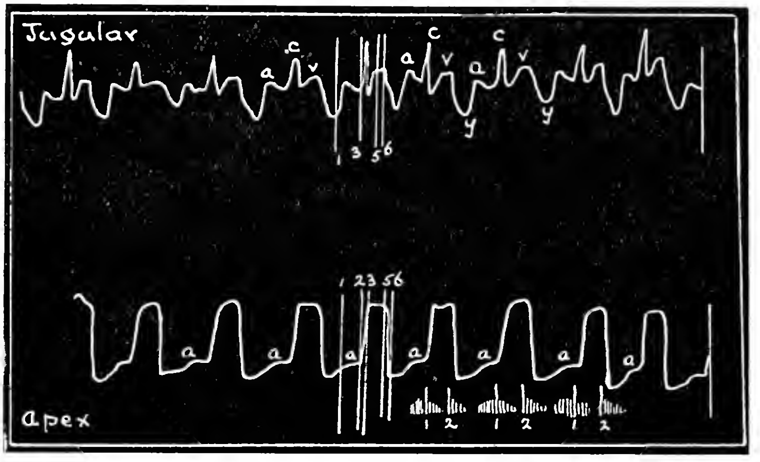


FIG. 231. Shows the auricular wave (a) in jugular and apex tracings. There is an increase in the time from the beginning of the auricular wave (a) to the large wave due to the ventricle in the apex tracing, and a corresponding increase in the a–c interval in the jugular tracing. Taken 1903. (Case 17.)

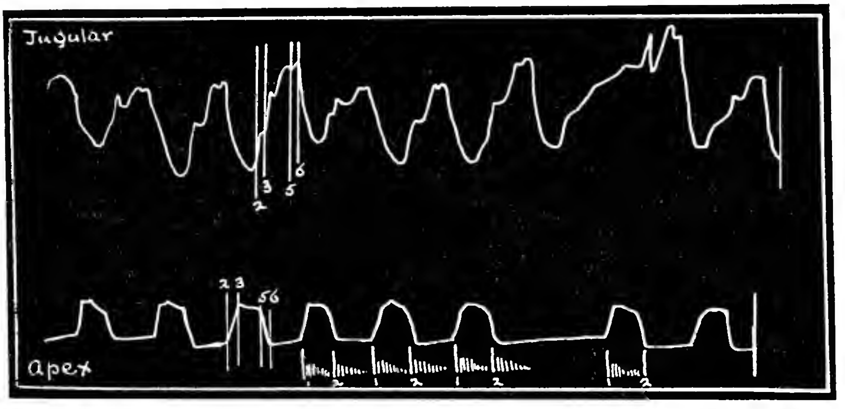


FIG. 232. With the inception of the nodal rhythm the heart is irregular, the auricular wave is gone from jugular and apex tracings (compare with Fig. 231), and the shading underneath shows a disappearance of the presystolic murmur. (Case 17, April 19, 1904.)

in the following week I found his heart perfectly regular, the auricular wave present in the venous pulse, the presystolic murmur, and the auricular wave in the apex tracing also present (Fig. 234). These conditions continued

until November, 1904, when his heart again became irregular, and all evidence of auricular systole again disappeared. From that date up to the present (1908) the heart has remained in this state, and on the numerous occasions when I have taken long tracings I have never yet found the heart regular for one moment (Fig. 238, Plate III).

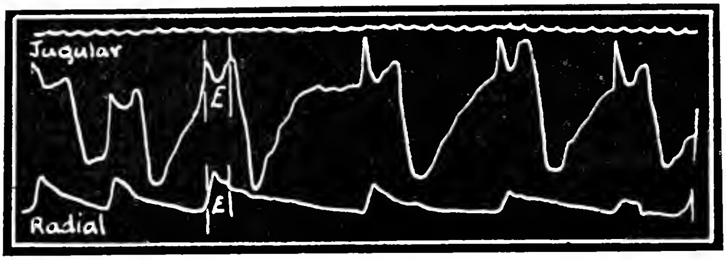


FIG. 233. Shows a slow irregular rhythm with the ventricular form of the venous pulse. (Case 17, April 19, 1904.)

FIG. 234. Shows a regular rhythm and the presence of the auricular wave in the apex and jugular tracings. (Case 17, April 26, 1904.)

CASE 18.—*Sudden inception of nodal bradycardia, lasting for about three weeks.*

Male, born 1852, a stout, healthy-looking individual. I had known him for about twenty-eight years, and had attended him at various times for trivial complaints, and in 1903 for an attack of erysipelas of the face. He had enjoyed good health, was getting fat and somewhat short of breath. On November 9, he was hurrying from the train to a football match, a mile distant from the station. As he approached the football field he was seized with pain across the middle of the chest, but as it was not severe he pushed on till he arrived at the field. He sat down, but the pain increased, striking into both arms, and his hands went white and cold. He felt as if he wanted to breathe deeply but could not. He endured the suffering for twenty minutes, and as it became worse, and he felt as if he would die, he was assisted off the field, put into a cab and driven to the station. He was

given some brandy, which made him sick. The pain gradually diminished, and he returned home by train; as he was better he walked home (about a quarter of a mile), but felt sick and short of breath. He went to bed, and one of my colleagues saw him and found his pulse between 30 and 40 beats per minute. I saw him next morning. He felt very weak; the pain was nearly gone, though it had kept recurring through the night. He had some pain if he took a deep breath. The pulse rate was 52, the heart's dullness extended from mid-sternum to 2 inches beyond the nipple line; apex beat faint in the fifth interspace; sounds clear and free from murmur. The superficial jugular vein was very full, but did not pulsate. The deep jugular was large, filling up during ventricular systole, and collapsing suddenly at the beginning of ventricular diastole. There was no sign of an auricular wave preceding the ventricular systole. The patient was kept in

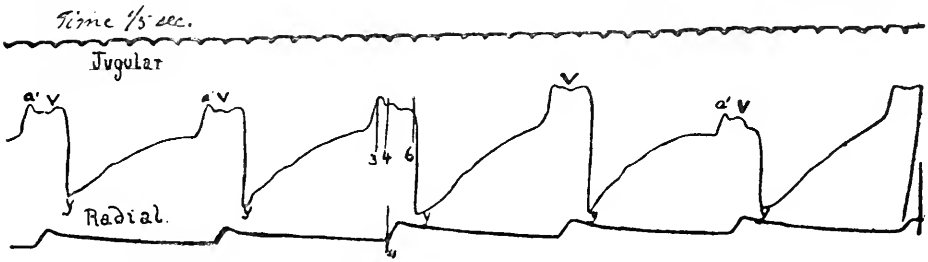


FIG. 235. Simultaneous tracings of the jugular and radial pulses. The jugular pulse is of the ventricular form, and shows the wave a' preceding by a short interval the time of the carotid pulse (perpendicular line 3). Pulse-rate, 40 per minute. (T.A., November 24, 1907.)

bed, and his condition did not undergo much change for the next fortnight, except that the pain gradually grew less till it finally disappeared and he was able to sit up. The pulse-rate varied, sometimes falling as low as 30, but never rising above 52. On November 24 a long tracing was taken with the ink polygraph, and the rate showed great uniformity; the rhythm was also quite regular. Fig. 235 is a small portion of the tracing taken on that day, and represents the same features as were present on November 10; the rate was 40 per minute, the rhythm regular, and the venous pulse of the ventricular form. When I next examined him on November 29, his pulse had increased in rate, with occasional intermissions. I had the greatest difficulty in getting a tracing of the jugular pulse; he had a very short fat neck. But imperfect as the tracings were, they showed a return of the auricular wave *a* to its normal period before *c*. From this date he gradually improved, and has been able to get about, though he is perhaps a little shorter of breath than before his attack. Tracings were taken from him in December, 1908, and on May 11, 1909. The venous

pulse was the same on both occasions, and showed an auricular wave, *a*, preceding the carotid wave, *c*, while the rate was 68 per minute; the rhythm was regular.

CASE 19.—*Inception of the nodal rhythm, the heart's rate at first not infrequent, but becoming slow with attacks of unconsciousness and epileptic fits. Recovery from these attacks with an increase in the heart's rate.*

Male, born 1838. I have known this patient intimately since 1894. He was a healthy, vigorous man up till 1907. He was a very heavy smoker, and for a great many years he smoked two ounces of tobacco and half a dozen cigars a day. I had occasion to examine him in 1906, and found his heart normal in rate and rhythm, though for some years he had been rather short of breath. I again examined him in February, 1907, and found that his heart was continuously irregular with the disorderly rhythm characteristic of the nodal rhythm. He was not conscious of the change, but there was a further increase in his breathlessness. He was still able to attend to business, and to play a game of golf. He lived some distance from me, and I did not see him again until October 11, 1907, when I was asked to see him with his medical attendant, Dr. O'Connor, to whom I am indebted for an excellent account of his many seizures. The history given was that his pulse had become very slow for some months, and that latterly he had been seized at times with attacks of unconsciousness. The pulse-rate on such occasions was found below 30 beats per minute. He was very weak and faint when I saw him, the pulse varying in rate from 30 to 40 beats per minute, and irregular with long pauses at times. During the long pauses there was often a small premature beat in the jugular (see *v'*, Fig. 236). The heart's dullness extended 1½ inches beyond the left nipple, and there was a soft blowing murmur at the apex. There was a small amount of albumen in the urine. The attacks of unconsciousness continued, and I saw him again in November, when the heart's condition was much the same. After this the pulse-rate increased, the attacks disappeared, and he went to Torquay in June, 1908, where he had a slight recurrence of his attacks of loss of consciousness. From this he recovered, and continued well till August 4, when after some effort he was seized with great breathlessness, and the attacks of unconsciousness recurred. These increased in number and severity, and for two whole days he was unconscious and deeply cyanosed. For some hours he passed from one epileptic seizure to another as if affected with uraemic convulsions. He also developed Cheyne-Stokes respiration. The pulse during these convulsive attacks was not perceptible. The severity of the attacks gradually lessened, and in the month of September his pulse-rate rose to 50 or 60 beats per minute. In

October he had a number of very transient fainting attacks. Dr. O'Connor described the attacks as resembling *petit mal.* Thus, while the doctor was talking to him, the patient's face would suddenly become pale, and consciousness would be lost for a brief period. During these attacks no pulse could be felt at the radial.

I saw him again on December 18, 1908. He was able to go about, and had been free from attacks for a few weeks. The pulse was rather slow, about 60 per minute, and irregular. The heart's dullness extended $1\frac{1}{2}$ inches beyond the left nipple, the sounds were clear, with a faint doubling of the first sound. There was no dropsy, and the urine was free from albumen.

On May 5, 1909, he was seen by Dr. John Hay, who took a long tracing with the ink polygraph. The rhythm was of the disorderly kind, charac-

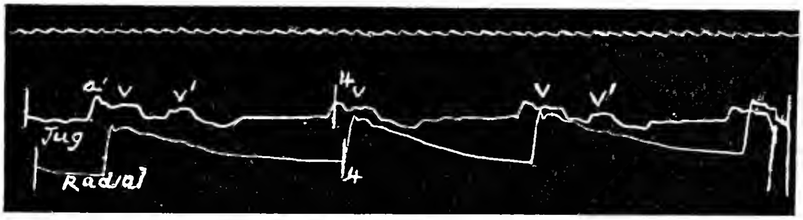


FIG. 236. Simultaneous tracings of the jugular and radial pulses. The jugular pulse is of the ventricular form. During the long pauses in the radial pulse, there are small premature beats, *v'*, in the jugular. The pulse-rate varied from 25 to 30 beats per minute. The patient was just recovering from a series of syncopal and epileptic attacks. (W.N., October 11, 1907.)

teristic of the nodal rhythm, and the jugular pulse was of the ventricular form. In August, 1909, the heart's action became very infrequent, and he was seized with attacks of loss of consciousness and epileptic convulsions, and he died in one of these attacks.

I had looked upon this case, at first, as an instance of Adams-Stokes syndrome, due to heart-block, but at the time I was puzzled to account for the fact that he had nodal rhythm prior to the onset of the attacks of unconsciousness and slow heart-rate, and also because of the nature of the jugular pulse, which was of the ventricular type. The tracing of the jugular has never been shown to be of this type in cases of heart-block. Case 18, which came under my observation in December, 1907, also puzzled me, and it was only when I began to analyse the tracings more carefully that the nature of the tracings from this patient dawned upon me, and that it became obvious that here we had an instance not of blocking of the stimulus between auricle and ventricle, but one of slow nodal rhythm producing anaemia of the brain.

CASE 20.—*Permanent nodal rhythm, bradycardia associated with mitral stenosis. Occasional attacks of syncope and convulsions (Adams-Stokes syndrome).*

Male, born 1865. When a soldier in India he had dysentery at the age of 20, syphilis at the age of 22. He had malarial fever in America at the age of 27. In 1894 he had the first attack of syncope. After lying up a week he went out, and in hurrying to avoid a cab he fell unconscious on the pavement, but quickly recovered. He consulted a doctor, who said his heart was affected. Two years later he was laid up with shortness of breath and swelling of the legs, and was treated for ' mitral disease '. He partially recovered, and had frequent attacks of weakness until the final

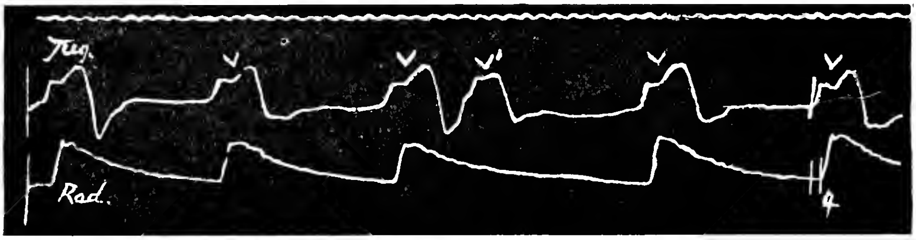


FIG. 237. Simultaneous tracings of the jugular and radial pulses. The jugular pulse is of the ventricular form, and the rate about 28 per minute. (M.M., November 9, 1908.)

breakdown occurred in 1905. He had been feeling ill for some years, but had worked hard, and had kept himself going on brandy, bovril, eggs, &c. He says his pulse was slow four years ago, and that it has remained so ever since.

In 1904 he began to have mild ' fits ' in which he lost consciousness and was slightly convulsed. From November, 1905, to April, 1906, he had a great number of fits, some severe with convulsions and cyanosis, others slight without convulsions. He had no attacks for a year, but he had a very severe one in April, 1907, and since then only three mild attacks. He had lived a life of hard work with frequent bouts of drinking.

The patient is tall, spare, and intelligent looking. The face is usually ruddy, with a faint duskiness. He walks slowly and carefully, and his gait is slightly ataxic ; if he hurries or gets excited he becomes giddy. He has a somewhat violent temper, and when in a passion his face becomes dusky and cyanosed. When lying down there is a large pulsation, seen in the deep jugular on both sides, heaving in the lower part of the neck, as in Fig. 237. It is very slow and synchronous with the apex beat.

The radial pulse is slow and deliberate, usually about 30 per minute, and usually quite regular. At times two beats are close together, and are

followed by a long pause. These coupled beats may appear at rare intervals or alternate with a single beat, or they may appear continuously for a short period. The apex beat is large and diffuse in the sixth interspace, and in the anterior axillary line. The heart dullness extends 1 inch to the right of the middle line and 8 inches to the left.

There is a rough blowing systolic murmur heard best at the apex and propagated towards the axilla. The second sound is clear and well struck and followed by a soft murmur. This diastolic murmur is heard only over a limited space at the apex, and is not always perceptible. It is, as a rule, faint and fades away.

A large number of tracings have been taken from this patient at different times and they always present the same features, the only difference being that sometimes the coupled beats are more frequent or are entirely absent. The jugular tracing shows one large wave occupying the whole time of ventricular systole.

The Tendency to Standstill of the Heart in Nodal Rhythm, with Notes of Two Cases

The tendency for long pauses in the heart's action in cases of nodal rhythm, such as those seen in Cases 17 and 19, has been a matter of interest to me for many years. These pauses may not produce any symptoms, but they may last long enough to produce anaemia of the brain with transient loss of consciousness, and even the more prominent phenomena of Adams-Stokes syndrome (Cases 19 and 20). The symptoms then resemble those of the more common condition, auriculo-ventricular block. I have been impressed by the fact that not a few cases of nodal rhythm die suddenly, and it seems that this tendency to long pauses shown in the figures may be the immediate cause of death, as in the following case. The proof of this view is not complete, but the following cases are also very suggestive.

Case 21.—*Nodal rhythm, with long pauses in the heart's action, producing attacks of unconsciousness and probably the death of the patient.*

Female, born 1854. I had known this lady for many years. She had a large goitre, and for the last few years of her life her heart was continuously irregular. She was not robust, but was able to attend to her household duties ; she was liable to attacks of palpitation, and had frequent attacks of syncope, which I could not account for at that time. On July 2, 1902, I was summoned to see her, and on my arrival I found her recovering from an attack of unconsciousness during which she had been convulsed. Her face had a deathly ex-sanguine appearance. She gradually recovered,

and her colour improved in the course of half an hour. On inquiry, I found that she was talking to her sister when she fell down in a faint and became convulsed for a few minutes.

When I examined her, the heart was acting irregularly but with fair strength. During the next few days her heart was irregular with long pauses, as shown in Fig. 239, taken on July 3, 1902. She sometimes lost

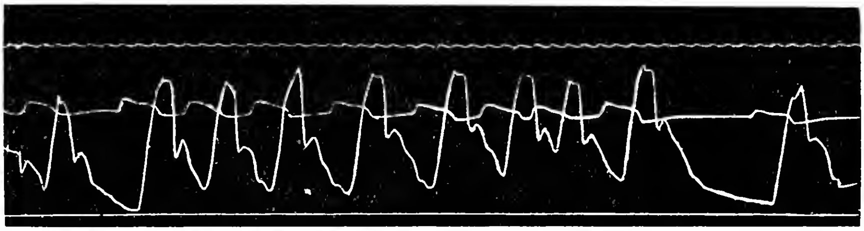


FIG. 239. Simultaneous tracings of the apex beat, and of the radial pulse. The rhythm is disorderly and characteristic of the nodal rhythm, with frequent long pauses. It was probably in consequence of these long pauses that the patient suffered from attacks of unconsciousness and convulsions. (M.B., July 7, 1902.)

consciousness for a few seconds, but I did not see her at those times. On July 7 I was again summoned to see her, but when I arrived she was dead. I was informed that she had again fainted, become convulsed, and then lay quite quiet.

FIG. 240. The jugular pulse shows a large auricular wave and an increased *a–c* interval (space *A*). (Case 22. 1892.)

CASE 22.—*Large auricular waves in the jugular and liver pulsation. Pre-systolic mitral and tricuspid murmurs. Auricular sound over the valves in the large veins. Sudden disappearance of all signs of auricular contraction, with the appearance of continuous irregularity in the heart's action. Sudden death.*

Female, born 1862; came under my care in 1891. She suffered from shortness of breath on exertion, and for some months before her death from severe attacks of pain striking from the front of the left chest down the inside of the left arm to the little finger, with great tenderness of skin over the area of pain after the attack had subsided. She had erysipelas in the

face in 1883 and 1885, and since then she has been short of breath, and with a tendency to swelling in the legs. The pulse was small, quick, and usually regular, but I occasionally detected an extra-systole, which Fig. 246 shows to be auricular in origin. There was a large pulsating swelling on

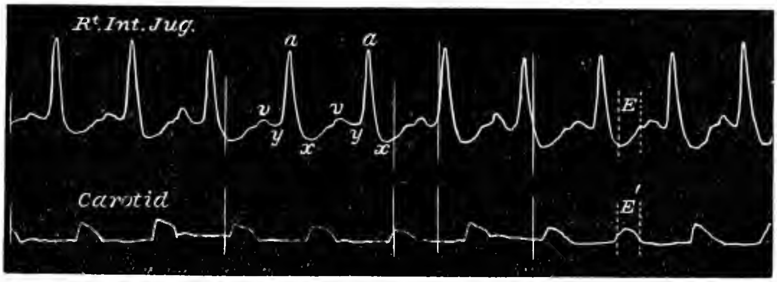


Fig. 241. Simultaneous tracings of jugular and carotid pulses. (Case 22. 1892.)

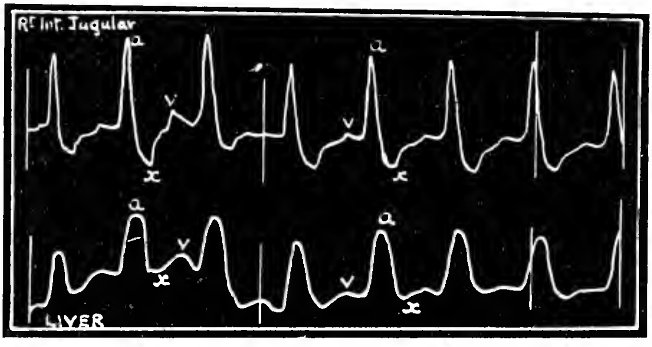


Fig. 245. Simultaneous tracings of jugular and liver pulses; *a*, auricular wave; *v*, ventricular wave; *x*, auricular depression. (Case 22.)

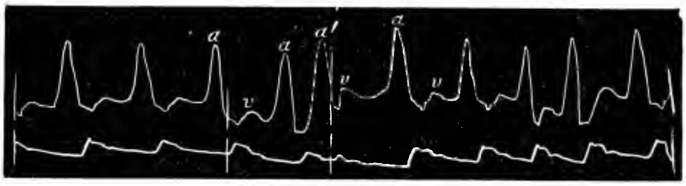


Fig. 246. Simultaneous tracings of the jugular and radial pulses, showing a large wave (a') due to an extra-systole of the auricle. (Case 22.)

either side of the neck near the sternal end of the clavicle, Figs. 240 and 241, but no distinct pulsation in the veins above these swellings. When compared with the carotid pulse there were seen to be two distinct movements, the one larger than the other, to each carotid pulse, and the larger movement could be observed to precede that of the carotid pulse (Fig. 241).

The liver could be felt pulsating just below the ribs, and the liver pulse was of the same character as the jugular (Fig. 245).

The area of cardiac dullness was increased, extending transversely one and a half inches to the right beyond the middle line. There was always present a long murmur, presystolic in time, heard best at the apex; another, shorter and rougher, of a different character, but corresponding in time, was heard best over the middle of the sternum. The latter murmur was occasionally absent at first, but ultimately became a constant phenomenon. There was also a murmur, systolic in time, heard at the apex. At the base the second sound was reduplicated. No murmur was heard in the carotids, but there was a distinct sound heard over the pulsating swelling in the neck synchronous with the pulsation and preceding the

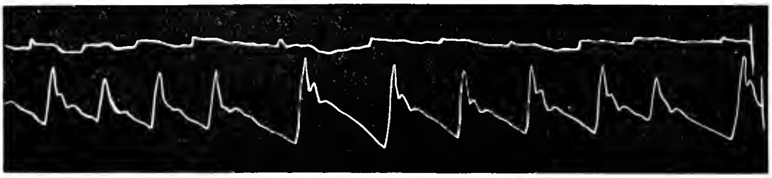


Fig. 247. Simultaneous tracings of a slight movement in the neck and of the radial pulse. The rhythm is now continuously irregular, with a tendency to long pauses. The tracing from the neck was taken from the same situation which gave the jugular pulse in the four preceding figures, and demonstrates the absence of any wave due to the auricle. (Case 22. 1895.)

first sound of the heart. This sound could also be heard under the clavicle and over Poupart's ligament—i.e. over the valves of the subclavian and femoral veins. The jugular and subclavian valves being competent, the jugular bulb became distended into a ball-like protrusion over the inner end of the clavicles, and the pulsation was conspicuous at the distance of ten yards.

These symptoms persisted, though the patient became gradually weaker and had severe attacks of angina pectoris on the slightest exertion. (Figs. 11 and 12 are drawn from her.) At 3 a.m., October 9, 1895, she awoke and was conscious that the beating in her neck had ceased. She called my attention to this next morning, and on examination I found that there was now no pulsation in the jugular bulb. Thus Fig. 247 was taken from her on October 9, 1895, and the receiver was held over the place where the jugular tracings Figs. 240 and 241 were obtained, and it will be observed that there is a complete absence of the auricular wave which is so marked in these tracings. In Fig. 247 there is only a faint movement due to the carotid, and possibly at the beginning of this tracing a slight ventricular venous pulse can be detected. Careful examination showed that the liver

pulse had entirely disappeared, as well as the mitral and tricuspid presystolic murmurs.

On October 13, 1895, on getting out of bed, she fell and died.

Before removing the heart at the post-mortem examination I injected water forcibly into the superior vena cava, and the water could not pass beyond the jugular valves, but distended greatly the jugular bulb. The report on post-mortem examination of the heart is as follows:—Marked tricuspid and mitral stenosis. There is dilatation of the apex of the left ventricle, pre-mortem clot in apex of right auricle. The series of sections of a.-v. bundle is good, and shows that in this heart the node at the commencement of the main bundle and the septal divisions were uncommonly well developed. Main bundle healthy. There are signs of great venous back-pressure in the capillaries and veins. The arteries in the neighbourhood of the bundle are not thickened.

I have dealt with this subject of nodal bradycardia more fully elsewhere[311].

APPENDIX V

Irregularities in Cardio-Sclerosis

The cause of the variations in the size of the pulse-wave is a subject that needs elucidation. There are different ways in which it may arise. The amount of blood in the heart at the time of the systole has little bearing in the cases to which I refer, inasmuch as there is usually plenty, the heart being somewhat dilated. Apart from this, the difference in the size of the pulse-wave depends on the strength of the ventricular contraction, which is determined by the contractile force of the heart muscle and the number of the fibres participating in the contraction. In a case of sinus irregularity, especially one of those occurring in young subjects, we find that, no matter how long or short the rest preceding the contraction, the beats themselves are of great uniformity (Figs. 73, 74, 79). On the other hand, in the irregularities of the nodal rhythm we find the size of the beat varies with the length of the preceding pause—the longer the rest, the bigger the beat. This implies that the recovery of contractile force is not so rapid as in the young with a sinus irregularity. This is the explanation adopted in § 179 to explain the pulsus alternans. But this explanation is not the only possible one. Muskens maintains that the smaller size of the beat is due to the fact that all the fibres do not contract, that there is a block preventing the stimulus for contraction from reaching all the fibres. He states that he has been able to produce the block experimentally in the frog's heart. On consideration, I have, however, adopted the view of Hoffmann and Wenckebach in regard to the pulsus alternans, though Muskens' views are applicable to other conditions of irregularity, and I have arrived independently at a similar conclusion. I refer to those instances in which there are varying sizes in the pulse-wave, and in which big waves may occur after a short period of rest, while very small waves may occur with a period of rest of the same duration. Here the smaller beats may be caused by some fibres of the ventricle failing to contract along with the general mass, or, as recent electro-cardiograms seem to show, the variation in the size of the beats may be due to the starting of the ventricular contractions from different places.

I have not been able to work out the matter to a satisfactory conclusion, but I have used it as a means of forming a prognosis, and I am inclined to

think this point will be of some value. The matter needs further consideration, and the different factors will be better appreciated by the study of typical irregularities, whose nature can be analysed with a greater degree of certainty. A good deal of preliminary work will have to be done to understand the different causes of variations in the strength of the beats. To that end, I quote here two cases that showed during life a somewhat bewildering variety of irregularities, and I demonstrate how these can be referred to their various causes with certainty. The citation of these cases also brings out certain important features due to cardio-sclerosis which must not be looked upon as exceptional, for I have seen a great many cases exhibiting the same features and due to the same causes. Briefly, these features are variations in the strength of the contraction, the size of the beat depending on the period of rest and the occurrence of extra-systoles.

Case 23.—Male, aged fifty-seven. Consulted me on May 1, 1905, complaining of weakness on exertion, a sense of great exhaustion and trembling of the legs. He first noticed the breathlessness three years ago on hurrying up a hill, when he had a severe attack. He felt fairly well until six months after, since which time the breathlessness is very easily provoked.

He is a life-long abstainer, and has led a steady, regular life. In his younger days his work entailed severe bodily effort, but for the last twenty years his occupation as mill manager has not demanded much exertion. Twenty years ago he had an attack of ‘ inflammation of the kidneys ’. He is a powerfully built man ; face greyish in colour, pulse rapid (86 per minute) and hard, the artery large and leathery in consistence. Heart is enlarged, dull to the nipple line. The sounds are clear and distinct ; the urine contains a large quantity of albumen. Blood-pressure, 210 mm. Hg. He was directed to take his food in small quantities, and to chew it thoroughly ; to have his bowels freely opened ; and he was put upon iodide of potassium. He improved wonderfully for a time, all the albumen disappearing from the urine, but he began to relapse. Abstinence from meat was tried and seemed to do him good for a short time, but he relapsed. The blood-pressure records were very confusing, sometimes falling to 145 and rising to 210, and this independent of drug or diet. There was no corresponding improvement in his condition with the fall of blood-pressure. When it was low he felt depressed, and sometimes felt very well when it was high. His pulse was usually alternating in rhythm, and the peculiarity was more marked when the pressure was high. When the irregularity had disappeared with lowered blood-pressure, it could easily be brought back by running up a flight of stairs. The difference in pressure between the beats was about 20 mm. Hg. —that is to say, if the radial pulse was obliterated by raising the pressure

in the cuff to 200 mm. Hg., and the air allowed to escape from the cuff, the large beats would be felt coming through at 190 mm. Hg., while the smaller would not be felt till the pressure in the cuff had fallen to 170 mm. Hg. (see Fig. 248).

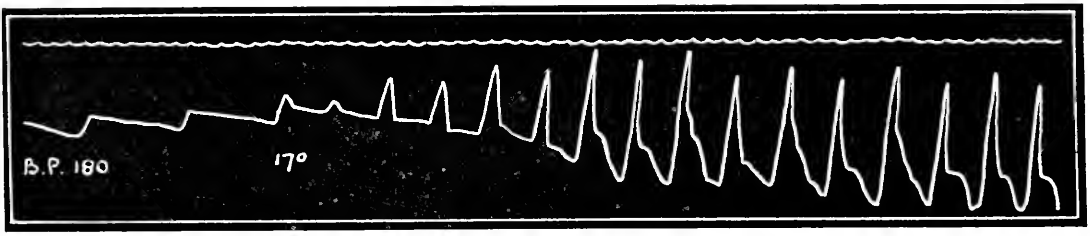


FIG. 248. Tracing taken from the radial artery after the air bag connected with the manometer had obliterated the pulse at a pressure of 190 mm. Hg. The air was allowed to escape gradually, and when the pressure fell to 180 mm. Hg. the sphygmograph received the stronger beats, and the smaller beats came through when the pressure fell to 170 mm. Hg. When the pressure in the air-bag was exhausted the pulse tracing showed rhythmical variation in the size of the waves—that is, the pulsus alternans.

Notwithstanding the alternating rhythm, the pulse rate was practically regular (Figs. 249, 250, also Figs. 168–170). In May, 1906, extra-systoles began to appear, and they gave a peculiar character to the tracing. Thus, in Fig. 249 the tracing shows a perfectly regular rate with a small and large

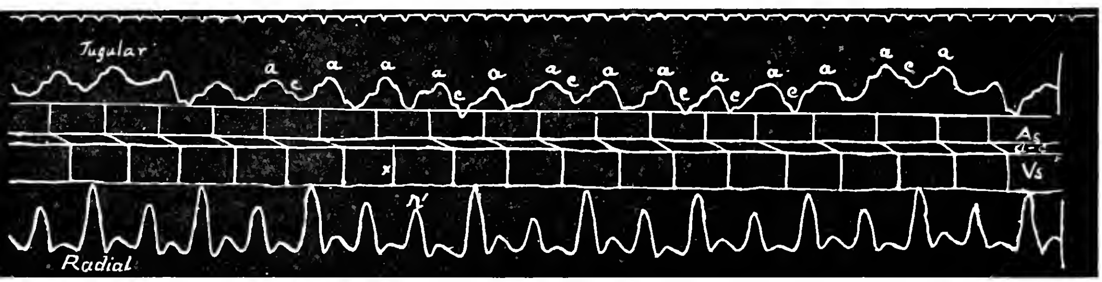


FIG. 249. Typical pulsus alternans with ventricular extra-systole r'. In the diagram it is shown to occur prematurely (downstroke $\times$) and independently of the auricular stimulus. After the extra-systole there is a longer pause, and the alternating character of the pulse becomes more marked. (Case 23.)

beat alternating, except in the centre, where there are two small beats in succession. On measuring the tracing it will be found that the second of the two smaller beats (r') occurs a little too early. The jugular tracing shows that the auricular wave (a) is perfectly regular, therefore r' is a ventricular extra-systole and after it there is a longer pause than normal, so that the beat after the pause is large, and the succeeding beat smaller than the other

beats for the reason explained in § 179. This increase of the alternating character of the rhythm is seen in Fig. 250. Here there are two extra-systoles, one as in Fig. 249 after the small beat, and the other after a large beat, and here also the character of the alternating rhythm is more marked after the extra-systole.

The patient also began to have attacks of angina pectoris, which I have described (Case 1, Appendix I). He became restless and disturbed at night, and the attacks of pain became more frequent, until he was put upon bromide of ammonium, when he began to sleep better, and the angina disappeared. Towards the end of 1906, Cheyne-Stokes respiration appeared (Fig. 6, Plate I), his nights became very restless, and he could only be relieved by opium and chloral. In January dropsy set in, the heart dilated, and his blood-pressure fell permanently to 150 mm. Hg. and under. The urine became scanty.

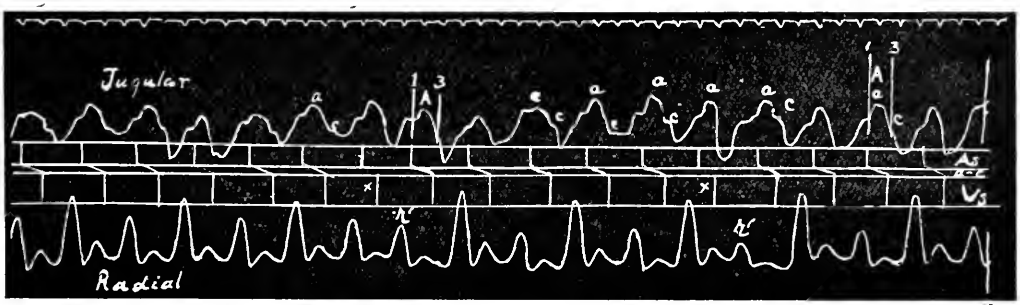


FIG. 250. Here there are two extra-systoles (r' r'), one after the small beat and the other after the large beat of the alternating rhythm. (Case 23.)

Various preparations of digitalis and other drugs were tried with little good results, and the patient died in March, 1907. Permission was only given to examine the heart, and the following is the report of the post-mortem appearance :—

Musculature of ventricles.—Hypertrophied, but the apical half of ventricle is fibrosed and dilated ; large pre-mortem clot adherent to the anterior wall of the left ventricle. Thickness of wall at base, 18–22 mm. ; over the fibrosed area, 6–8 mm. The musculature at the mouth of the superior vena cava is hypertrophied. Taenia terminalis is hypertrophied, and under the microscope shows many fibres atrophied and fibrosed.

Valves and orifices.—Mitral cusps thickened ; tricuspid, pulmonary, and aortic healthy. Auriculo-ventricular orifices smaller than normal, due to tonus or contraction of the musculature of the base.

Arteries.—Patches of atheroma in aorta, especially at orifices of coronary arteries. Intense endarteritis of left coronary artery, diameter 6 mm.,

lumen 2·5 mm.; the interior is especially thickened. The right coronary artery is not so much affected. All the arteries of the heart above 1·5 mm. in diameter are affected if they lie *outside* the musculature of the heart; if surrounded and supported by the musculature, less affected. The anterior inter-ventricular artery was most affected, while the artery to the a.-v. bundle from the right coronary was more like a needle-prick.

Remains of primitive cardiac tissue.—Sino-auricular node, less musculature (more fibrosed) than in health, still not marked. The a.-v. bundle is normal in size, and its fibres and cells are normal.

Case 24.—Male, aged seventy-two. I have known the patient for over twenty-five years as a steady, sober, and industrious man. He worked at his occupation as an engineer up to within a year of his death—though in later years he did not do much laborious work. He had a slight attack of hemiplegia in December, 1906. He consulted me in June, 1907, because he passed blood in his urine. Except for being rather short of breath, he felt fairly well. He looked a hale old man. His radial arteries were large and tortuous, pulse full, seemingly regular. His heart's dullness extended to the nipple line. The sounds of the heart were clear except for a musical murmur, systolic in time, heard over the whole heart region, but loudest in the aortic area. It varied distinctly in loudness, a loud murmur alternating with one less loud. When I took a radial tracing it showed a well-marked pulsus alternans. He visited me several times. The urine became quite clear, but the heart's condition continued, and extra-systoles were sometimes very frequent, and gave to the pulse the appearance of extreme irregularity (Figs. 243, Plate III, 244, Plate IV).

Early in August he complained greatly of attacks of bad breathing in the night. He would go to sleep quite quietly and then awake suddenly with a sense of suffocation, and sit up in bed breathing heavily. After half an hour he would feel easier, but could not lie down, and had to be propped up in bed. These attacks occurred on several occasions, until they disappeared in September, when his legs began to swell, and he expectorated quantities of blood-stained mucus and small clots of blood. His heart's dullness extended two inches beyond the nipple line; the veins of the arm became full. Venesection was tried, but with little good result, and he died in October.

An analysis of the tracings taken shows that the irregularity which looked so hopelessly confused was due to the mixture of the pulsus alternans with extra-systoles. Thus, Fig. 242, Plate III, shows exactly the features described in Fig. 250, where there are several extra-systoles (r') after the large beat and one after the small, with an increase in the alternating character of the rhythm after the extra-systole. Sometimes the extra-systole appears

regularly after each small beat, and the pulse has the appearance of what used to be called a trigeminal pulse, but this is simply due to a big beat after the long pause followed by a small normal beat, which is in turn followed by a ventricular extra-systole (Figs. 243, Plate III, 244, Plate IV). The jugular tracing is due to the auricle and varies at times in rhythm, so as to give rise to the suspicion of auricular extra-systoles at *ax*. The patient being unable to hold his breath, the jugular pulse varies in size with the respiratory movements. These movements are recorded in Fig. 244, Plate IV. The respiratory movements render it somewhat difficult to be sure of the beginning of the auricular systole, and I have endeavoured in the intercalated diagrams to show the relative time of the auricular and ventricular systoles, and the time the stimulus took to pass from auricle to ventricle. Except in Fig. 243, the slanting line in the middle space represents the *a–c* interval ; in Fig. 243 it represents the interval between the auricular systole and radial pulse. It will be seen that the duration of the interval varies, suggesting that the cardio-sclerosis from which the patient suffered also affected the a.-v. bundle—a suggestion which receives some support from the post-mortem examination.

Report of the post-mortem examination of the heart :—

The mitral and tricuspid valves show no pathological changes. The auricles are not markedly dilated ; the coronary sinus is filled with post-mortem clot. The ventricles are not dilated, except at the apical part of the left. Both coronary arteries show a thickening of their coats, with dilatation, the left more than the right, and the anterior interventricular branch of the left most affected. At the apex of the left ventricle is an area showing intense fibrosis with deposits of premature clot, but microscopic sections show the Purkinje and inner muscle-layer healthy.

The a.-v. node and bundle show a fibrosis, not intense, and also a stretching of the bundle, as if the pars membranacea had been stretched. There is no sign of cellular change in the bundle or node, except the predominance of the fibrous tissue over the muscle tissue—the muscle-fibres, instead of reticulating, being stretched and parallel.

APPENDIX VI

The Effects of Digitalis on the Human Heart

In Chapter XXXIV I dealt with this subject, and here I cite a few cases to illustrate further the importance of this line of investigation, and to call attention to the varying effects of digitalis on different heart-lesions. I wish also to impress upon hospital physicians, by these illustrative cases, the great opportunities they have of demonstrating by such observations the effects of remedies. The experiences I detail here were obtained in the course of my daily work as a general practitioner, and from among my private patients. The difficulties of sufficiently precise observation were insuperable, so that of necessity my work lacks the precision of a physiological experiment—a precision, however, that could easily be attained were the patients under continual observation in a hospital ward.

This line of investigation has, I am glad to say, been adopted by several physicians, and John Hay [99], A. G. Gibson [96], Hewlett [100], Gossage [97], Lewis [101], and Guilleaume [98] have demonstrated equally striking results from the administration of digitalis.

In the interpretation of the phenomena given here, I have been greatly assisted by Professor Cushny, whose knowledge of the physiological action of digitalis is unrivalled.

The following case is important, as it shows the rhythm of the heart changing from the normal to the nodal rhythm, and back again to the normal, and the difference in the behaviour of the heart to digitalis under the two rhythms.

Case 25.—Female, aged sixteen. Seen first on March 21, 1903. Has had rheumatic fever, and for some years has suffered from breathlessness and palpitation on exertion. This has increased lately, and there is slight oedema of the legs. There is marked pulsation of the veins of the neck of the auricular type (Fig. 251). The pulse is small, regular, 86 per minute. There is great heaving of the left chest with the movements of the greatly enlarged heart. The apex beat is large and diffuse, and felt in the sixth interspace and in the anterior axillary line. There is a loud, rough systolic murmur heard over the whole heart and round to the back, but loudest at the apex. With rest and digitalis she rapidly improved. She broke

down again, and a note on January 9, 1904, states that the abdomen is greatly swollen, the liver enlarged, the legs oedematous, and the urine scant. The radial pulse is small, soft, and rapid, 126 per minute, while the jugular pulse is of the ventricular type. Under digitalin granules she again improved, but in February the granules were stopped, and she speedily

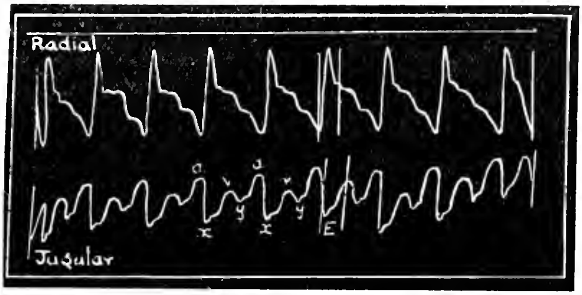


FIG. 251. This and the following seven tracings are from the same patient. Here the pulse is regular, and the jugular pulse is of the auricular type. (Case 25, March, 1903.)

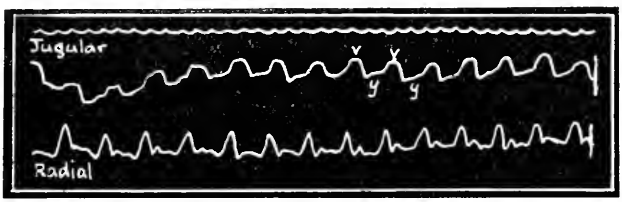


FIG. 252. The jugular pulse is now of the ventricular type. (Case 25, March 10, 1904.)

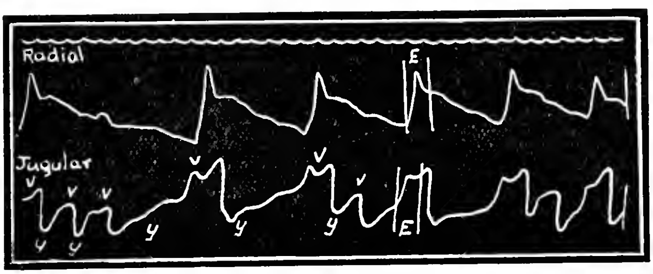


FIG. 253. The jugular pulse is still of the ventricular type, but under the action of digitalis the radial has become slow and irregular. (Case 25, March 18, 1904.)

broke down again. On March 10, 1904, the condition was similar to that described on January 9, the radial and jugular pulse-tracings being shown in Fig. 251.

She was prescribed digitalin granules, one per day. They speedily took effect, and on March 18 the pulse had become slow and irregular (Fig. 253). The urine had greatly increased in quantity, the abdomen and

liver had diminished in size, and all signs of dropsy had gone. The digitalis was continued till March 28, one granule being taken every second day, and she continued in fair health, the pulse still slow and irregular, as shown by Fig. 254, which was taken on March 26.

The digitalin was stopped on March 28. Four days after stopping it the pulse had increased to 85 per minute, though she felt still fairly well.

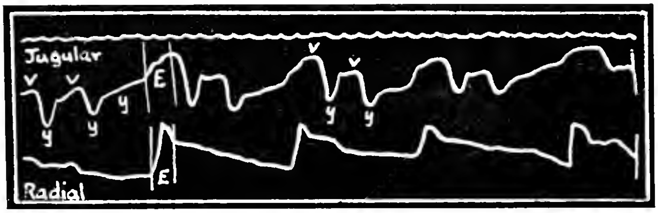


FIG. 254. Shows the characteristic effect of digitalis being maintained. (March 26, 1904.) The jugular tracing shows the nature of the arrhythmia, and the coupled beats resemble the tracings in Figs. 153, 157, 159, and 160. (Case 25.)

On April 5—that is, eight days after stopping the digitalin—the rate of the heart had increased to 120 beats per minute, the pulse had become small and weak, and the jugular distension had increased (Fig. 255). The other signs of heart failure were beginning to show themselves. She was again put on digitalin, one granule per day. On April 9 the pulse was still

FIG. 255. Eight days after stopping the digitalin, failure of compensation again set in, the pulse here being 120 per minute, and the jugular pulse being still of the ventricular type. (Case 25, April 5, 1904.)

120 per minute. On April 11 it was 130, on April 14 it had again become slow and irregular (Fig. 256).

The digitalin was again stopped, but as the pulse began to increase in rate, on April 17, one granule per day was prescribed. The patient continued in fair health, but as the pulse did not slow down satisfactorily, on May 1 I doubled the dose. I did not take any further tracings till May 14, contenting myself with watching for the slowing of the pulse. Finding it did not yield as before to the increased doses of digitalin, I took tracings

on this day, and found a perfectly regular radial pulse, while the jugular pulse had completely changed its character, being now of the auricular

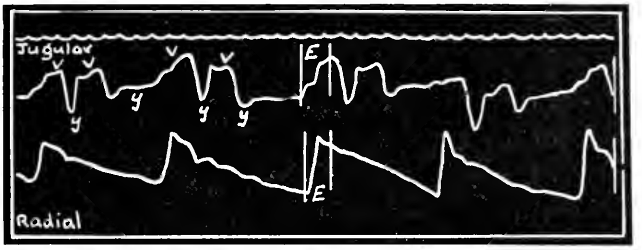


Fig. 256. Nine days after beginning the digitalis the characteristic effect is reproduced. (Case 25, April 14, 1904.)

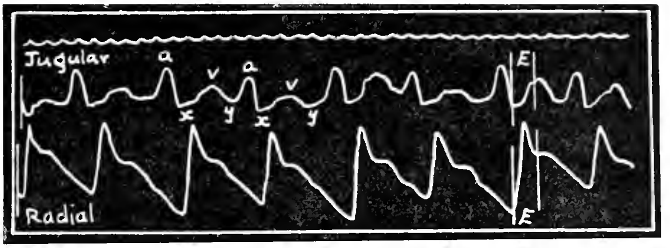


Fig. 257. With continued use of digitalis the auricles resume the inception of the rhythm of the heart, as shown by the fact that here the jugular pulse is of the auricular type. (Case 25, May 15, 1904.)

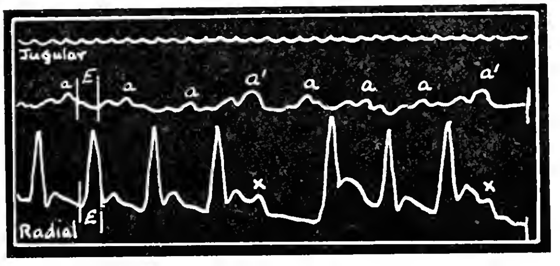


Fig. 260. Shows a jugular pulse of the auricular type with the occasional occurrence of a ventricular extra-systole. The auricular waves (a and a') occur at regular intervals, while the small waves ($\times$) in the radial occur prematurely. The larger size of a' is due to the fact that when the auricle contracts the ventricle is already in systole, and therefore cannot receive the auricular contents, which are thus sent back into the veins, producing this larger wave. (Case 25, January 2, 1905.)

type—that is, the auricle had again resumed its normal action and the heart chambers contracted in their normal sequence. Occasionally, for a short period, it would show a slight alternating rhythm, as in Fig. 257.

The digitalin was stopped, and the patient continued in fair health for some months. The jugular pulse continued of the auricular type, and the pulse was quite regular until her death in December, 1905, except during a short period shortly to be described. On December 18, 1904, she was again beginning to get oedema of the legs, and the abdomen began to swell, and she was very breathless. The pulse was small, soft, and rapid, 110 per minute, and the jugular pulse was still of the auricular type. She was prescribed one granule of the digitalis per day. No improvement had taken place by December 27, when she was ordered to take two granules per day. By January 2, 1905, the rate had fallen to 80, as a rule quite regular, but occasionally an extra-systole of ventricular origin would occur (Fig. 260). Sometimes for a short period these ventricular extra-systoles would appear after every second beat. The digitalis was stopped, and the arrhythmia disappeared.

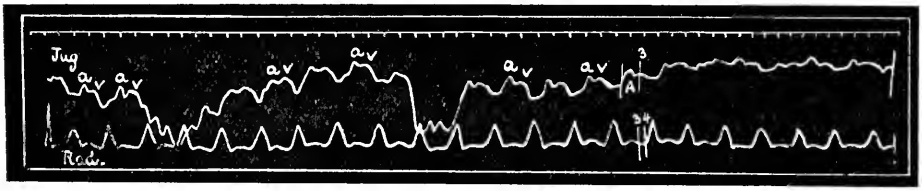


Fig. 262. Simultaneous tracings of the jugular and radial pulses. The jugular pulse is of the auricular type and the distension of the veins obscured the carotid. (Case 26, April 22, 1907.)

Case 26.—Male, aged eleven, seen by me on April 26, 1907 ; complained of weakness, shortness of breath, and palpitation. He had rheumatic fever at the age of seven. His pulse was rapid (110 per minute), small, and regular (Fig. 259). The heart was enlarged, the apex beat diffuse, extending one inch beyond the nipple line. There was a long, loud systolic mitral murmur. There was only a faint pulse in the distended jugular, and I had some difficulty in getting a tracing (Fig. 262). It was of the auricular form, and the *a–c* interval (space *A*) was rather greater than would normally appear with so rapid a heart-rate. I prescribed digitalin granules, one granule per day, and after taking ten granules he became very sick and vomited, and his pulse became slow and irregular. The granules were at once stopped, and after the sickness subsided the boy felt much better and remained in a fair state of health up to the last time I saw him, in November, 1907. I took a large number of observations when the heart was slow from the effects of digitalis, and the irregularities were all due to the same cause—the dropping out of ventricular systoles as shown

in Fig. 264. I have constructed a diagram showing the character of the irregularity, and it can be seen that the auricle pursues a regular course; that the *a–c* interval varies, being shorter after a pause and lengthening until a beat drops out (Fig. 269).

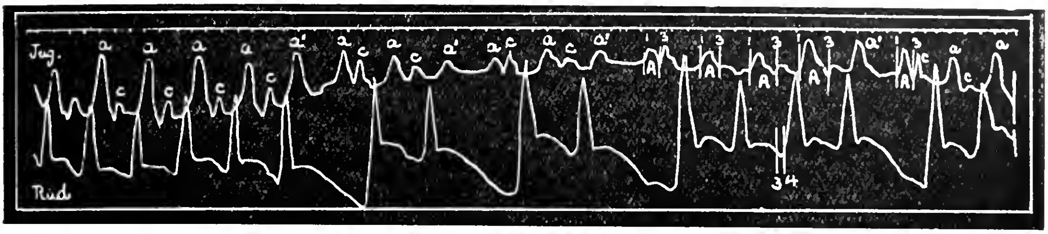


FIG. 264. The auricular wave, *a* and *a′*, is regular. The long pauses in the radial tracing are due to dropping out of the ventricular contractions—the stimulus from the auricle being blocked at the a–v bundle (see diagram, Fig. 269). Note the variation in the *a–c* interval (spaces *A*). (Case 26, April 26, 1907. After 10 digitalin granules.)

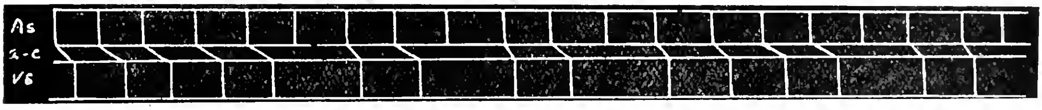


FIG. 269. Diagram showing the auricular and ventricular systoles from Fig. 264. Note the lengthened *a–c* interval before and the shortened *a–c* interval after the dropping out of the ventricular contraction. (Case 26.)

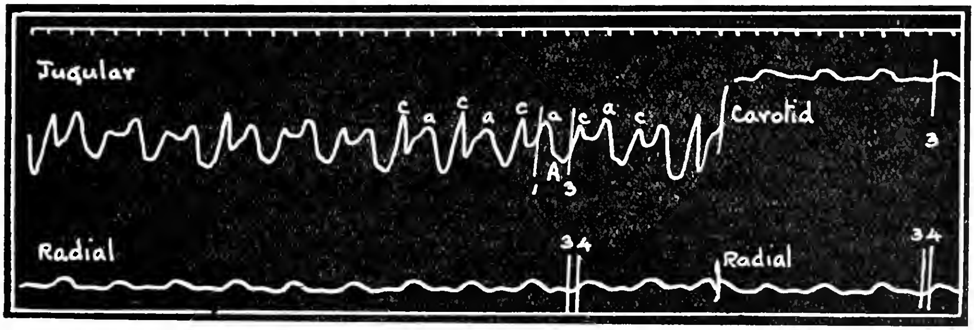


FIG. 270. The jugular tracing shows a wide *a–c* interval (space *A*). (Case 27. Before digitalis.)

CASE 27.—Male, aged twenty-five, consulted me for a stiffness and swelling in sundry joints, wrists, ankle, and knee, on May 4, 1906. The heart was rapid in its action, 120 per minute; slightly enlarged with systolic mitral and tricuspid murmurs. There was marked pulsation in the neck, of which Fig. 270 is a tracing. The movement due to the carotid was always large, and forms a marked feature (wave *c* in all the tracings). In the course of the next fortnight there gradually developed double aortic

murmurs. By May 23, under treatment, he had gradually improved, the rate of the heart falling to 90 beats per minute.

As the *a–c* interval, space *A*, in Fig. 270, showed a delay in the function of conductivity, I reasoned that the carditis had probably affected the a.-v. fibres, and had depressed the function of conductivity. I administered digitalin granules, one to be taken three times a day.

I kept him under observation, but could detect no change in the heart's action until May 30, after he had taken nineteen granules. On this date I found the pulse at times very irregular. Fig. 271 is a tracing, showing the nature of the irregularity. Between the jugular and radial pulses I have intercalated a diagram illustrating the events in the tracing from the neck. It will be noticed that before a ventricular beat drops out

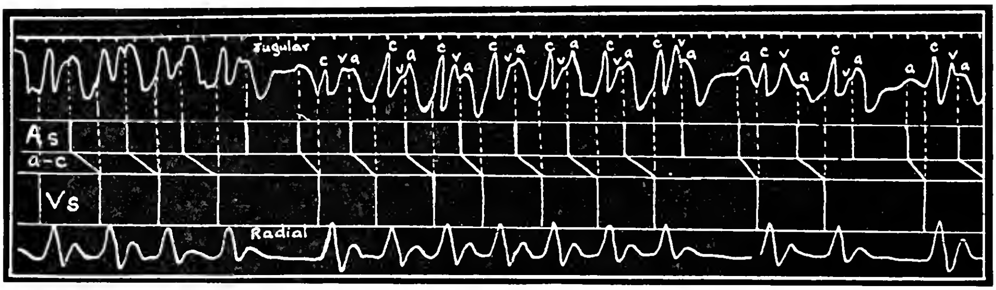


Fig. 271. After taking nineteen digitalin granules the pulse became irregular, which the intercalated diagram shows to be due to dropping out of the ventricular systoles—a mild form of heart-block. (Case 27.)

there is a gradual lengthening of the *a–c* interval, and that the dropping out of the ventricular systole is manifestly due to an increased depression of the conductivity of the fibres joining *a* and *v*—that is to say, the stimulus from the auricle is blocked before it reaches the ventricle. I stopped the digitalin, and a few days later all signs of irregularity had disappeared. The patient himself was conscious when his heart was irregular, and I remarked to him that the irregularity had gone ; he replied, 'I can bring it back.' I asked him how he could do so, and he said, 'By swallowing.' I asked him to swallow, and he did so, and immediately I detected long pauses in his pulse, while, on auscultation of the heart, no sounds were heard during the pauses. I took a large number of tracings for an hour and a half, during which time he swallowed forty or fifty times, and the alteration in the pulse-rate never failed to appear. The characteristic changes are seen in Figs. 258, Plate IV, and 259, Plate IV. After swallowing there are each time three regular beats, then the pulse slows in the manner shown in the tracings. After two or three slow beats the

rate of the heart increased for six or seven beats, then gradually slowed in the manner shown in the latter part of the tracings. Occasionally during the secondary slowing one ventricular systole would drop out, as is shown in Fig. 259, Plate IV. In Fig. 259, Plate IV, I have intercalated a diagram which shows the nature of the arrhythmia, and it can there be seen that the long pauses in these tracings are preceded by an increase of the *a–c* interval, just as happened when the patient was under the influence of digitalis (Fig. 271), and that the dropping of the ventricular systole was due to a block of the stimulus from auricle to ventricle. The numbers given under the radial tracings in Fig. 259, Plate IV, represent tenths of seconds, and from these numbers the manner in which the rate of the pulse varies can better be realized.

The susceptibility of the heart to the act of swallowing continued for a week, then entirely disappeared. There is no doubt it arose through reflex stimulation of the vagus induced by the act of swallowing. The analogy between the effects of digitalis and of reflex stimulation of the inhibition by swallowing appears worthy of note, as it indicates that the action of the drug here is exerted through its effects on the inhibitory centre, and not through the changes it induces in the heart muscle directly. Digitalis, as is generally known, affects the vagus centre and also the myocardium, and it is often difficult to determine which is the factor in its therapeutic effects. This case, taken along with Case 28, seems to give valuable data on this question.

The cases I have quoted here and in Chapter XXXIV present the more common effect of digitalis on diseased hearts. Other cases crop up that show different changes, some of which are capable of being analysed, so that one can tell with fair certainty what is happening, though the manner in which the phenomena are produced remains obscure. These cases are nearly all due to rheumatic affection of the heart. The following case showed a series of peculiar symptoms, and I select the most significant of these. It is impossible here to give fully the whole of the observations in this case, for verbal description is of little use, the tracings alone demonstrating the changes, and I have taken as many observations on this patient as would fill a goodly volume. I am also unfortunately unable to give minute particulars as to the quantity of the drug that produced the symptoms, for the patient was not confined to bed, and, living some distance from me, visited me at irregular intervals. Being the wife of a working man she had her household duties to perform, and when feeling better did not trouble to come to see me. As the improvement in her condition always coincided with the changes in the heart's rhythm, I was not able

to note the beginning of the changes and the quantity of the drug that induced them.

The drugs used in this case were the digitalis, squill, and calomel pills, digitalin granules, and the tincture of strophanthus. So far as the heart's irregularities were concerned, each form of medicine produced the same result. The patient preferred the digitalis, squill, and calomel pill, as it was the speediest in diminishing the dropsy by inducing an increased flow of urine, and a little diarrhoea, and the appearance of the diarrhoea was always accompanied by improvement in the breathing.

When not taking any drug the heart was quite regular in its rhythm until the last month of these observations, when the nodal rhythm became established. Under the influence of the digitalis or strophanthus the following irregularities appeared :—

(1) Nodal rhythm (transient).

(2) Slowing of the auricles and ventricles, the whole heart participating in the slow action.

(3) After a long pause the heart's contraction started feebly, and increased in strength with each succeeding beat (staircase phenomenon).

(4) Extra-systoles.

The *a–c* interval when the heart was not under the influence of the drug was always greatly increased, and the digitalis did not increase the delay, as in the cases already quoted. During the long pauses the *a–c* intervals became greatly shortened. As there was in this case a marked presystolic murmur, the relationship of this murmur to the first sound varied with the length of the *a–c* interval, sometimes being separated from the first sound, sometimes running up into it, and sometimes not being distinguishable from it (see Fig. 265, Plate V). During the nodal rhythm the presystolic murmur disappeared.

Case 28.—Female, aged twenty-eight. Consulted me April 24, 1907, complaining of swelling of the abdomen, shortness of breath, and palpitation. She began to feel ill in November of the previous year ; she had rheumatic fever fifteen years previously. The face is dusky, the breathing laboured, the legs and abdomen swollen. She passes little urine. There is rapid pulsation in the veins of the neck—two to each radial pulse (Fig. 272)—the pulse is small and regular, blood-pressure 100 mm. Hg. A thrill systolic in time can be felt over the upper part of the chest. The movement of the heart farthest to the left is three inches beyond the nipple, and shows an indrawing during ventricular systole (giving rise to an inverted cardiogram). The heart's dullness extends half an inch to the right of the sternum. At the apex there is heard a presystolic murmur, a diastolic murmur, and a reduplicated

second sound. At the base there is a loud, rough murmur systolic in time, heard also over the carotid. There is also a slight diastolic murmur at the aortic area. Over the heart's dullness inside the nipple there is another murmur systolic in time, and while listening one could imagine that two hearts were working, a series of sounds being heard immediately under the stethoscope, while another series of sounds could be heard faintly and seemingly at a distance.

Sometimes when the heart was regular but slow, a confusing change took place. During the diastole of the heart there was a sudden increase in the intensity of the diastolic murmur, in fact a mid-diastolic murmur followed after a pause by the first sound. When I drew the position of

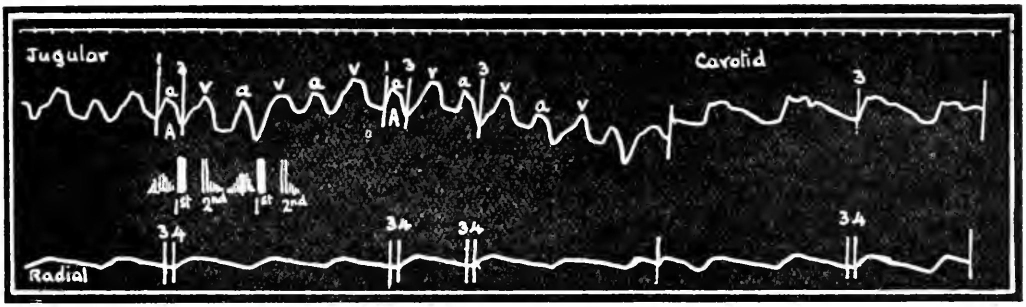


Fig. 272. Shows two pulsations in the jugular tracing (a and v) to one radial pulse. The a–c interval (space A) is increased. The shading shows the sounds of the heart and the murmurs present at the apex, viz. a presystolic murmur separated by a brief interval from the first sound, a reduplicated second sound followed by a diastolic murmur. These features of the jugular pulse and sounds and murmurs were always present when the patient was not under the influence of digitalis until the final establishment of the nodal rhythm as shown in Fig. 268, Plate V. (Case 28.)

the murmur under a tracing I found it corresponded to the position of the auricular wave in the jugular tracing (Fig. 272), and no doubt it was due to the auricular systole. When the heart became very slow, because of the digitalis, the longer rest gave the a.-v. fibres time to recover and the murmur was heard nearer and nearer the first sound, and sometimes it ceased to be heard. This only happened when the heart became very slow and the auricular wave in the jugular approached quite close to the carotid. These changes in the sounds and position of the auricular wave are seen in Fig. 265, Plate V. I am aware that this explanation may appear fanciful, but it is the outcome of long and patient study on the part of myself and some of my colleagues. My colleague, Dr. Crump, who is a very skilled auscultator, verified, after a long examination, these changes in the position of this auricular systole, and when shown the tracings which were taken

while he was listening, agreed to the place of the murmur indicated by the shading in Fig. 265, Plate V. I dwell at length upon this on account of the views that are held by some able clinicians that the presystolic murmur in mitral stenosis is not due to the auricular systole.

The patient was put on the digitalis, squill, and calomel pill, one taken three times a day. On May 1, after taking eighteen pills, she was passing more urine. She felt much better on the 8th—the swelling had gone from the abdomen and the legs were less in size. On the 15th the swelling had all gone, she had a little diarrhoea and felt sickly, but breathed easier and was able to walk better. The heart was slow and irregular. On examination there was only one wave visible in the jugular, and when recorded it was found synchronous with the carotid pulse (Fig. 261, Plate IV). There was no presystolic murmur, but only a long diastolic murmur at the apex, diagrammatically represented in Fig. 261, Plate IV. The tracing shows the jugular pulse to be of the ventricular type, i. e. the heart has taken on the nodal rhythm. The pills were stopped, but the heart was still irregular on the 19th. On the 26th it was regular and the jugular pulse double waved as in Fig. 272. She felt better, but the legs and abdomen were swelling again. Digitalin granules were prescribed, one three times a day. On the 31st the pulse became irregular and tracings taken on this day and on June 2 showed that it was due to the slowing of the whole heart (Figs. 273, and 263 Plate IV). On June 4 the heart had again taken on the nodal rhythm (Fig. 261, Plate IV), which persisted until the 17th, when the auricular contractions appeared and the heart showed frequent pauses. She was taking one granule of digitalin per day until the 23rd, when it was stopped, and on the 28th the heart was found rapid and regular and the pulsation in the veins double waved, as in Fig. 272. Up till November 4 these reactions due to digitalis continued to appear; sometimes the nodal rhythm would appear and sometimes the long pauses as in Figs. 273, and 263 Plate IV, and occasionally extra-systoles as in Figs. 266 and 267, Plate V. A few days after stopping the digitalis the heart invariably became regular. On November 6, after digitalis treatment had ceased, the heart assumed the nodal rhythm (Fig. 268, Plate V) continuously, beating rapidly, but it could be slowed by digitalis or strophanthus.

Interpretation of tracings.—The tracings showing the nodal rhythm (Figs. 261, Plate IV, and 268, Plate V) call for no special description, as they resemble the tracings of the ventricular venous pulse so fully described elsewhere. The only point which seems novel is that when it began under the influence of digitalis (Fig. 261, Plate IV) it was a slow

rhythm resembling in some respects the cases of nodal bradycardia (Appendix IV). On the other hand, when it started independently of digitalis the heart's action was rapid, as is usually the case.

In Fig. 273 there is a long pause in which the whole heart stands still; during these pauses no sound could be heard and the tracings show the auricles arrested as well as the ventricle—differing thus from heart-block. I observed that after the long pause the radial beats were at first small, then gradually increased in size, but as the pulse was small and soft the ink polygraph did not show the beats well; I therefore took several tracings with the Dudgeon sphygmograph, of which Fig. 263, Plate IV, is a good instance. Here the gradual increase of the radial pulse after the long pause is very evident. The pauses lasted sometimes from three to four seconds. Thus in Fig. 263, Plate IV, one pause lasts fifteen-fifths of a second, while

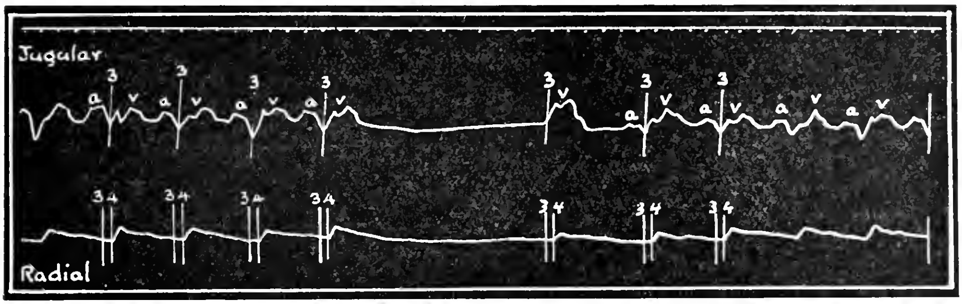


Fig. 273. Shows the temporary arrest of the whole heart from digitalis. (Case 28.)

the other lasts nineteen-fifths of a second. This standstill of the whole heart is probably due to vagus stimulation. The staircase phenomenon after the pause has been shown experimentally to occur after a vagus standstill of the whole heart, and, according to Gaskell, arises in two ways: (1) From exhaustion of contractility—the vagus stimulation depresses all the functions and their restoration is gradual, that of restoration of contractility being shown by a gradual increase in the strength of the beat. (2) From depression of conductivity, the stimulus for contraction not spreading throughout the whole heart, but reaching at first a limited number of fibres, and gradually reaching more and more until they all respond.

The pauses were not always so long as in Fig. 263, Plate IV, and the heart would beat slowly for a short period. When this happened, the relation of the auricular systole to the ventricular underwent an interesting change. As I have already remarked, the *a–c* interval was always increased in this patient, but digitalis did not interfere with the conduction of the stimulus

from auricle to ventricle. When the heart acted slowly the a.-v. fibres obtained a long rest, with the result that the *a–c* interval gradually diminished until it was scarcely perceptible. Fig. 274 shows the carotid and radial pulses. The carotid is taken from under the right jaw, but the wave (*a*) due to the auricular systole is present in the tracing. When the heart was beating at the more rapid rate, the *a–c* interval (spaces *A*) was nearly two-fifths in duration, whereas when beating at the slower rate it became less than one-fifth in duration. This is brought out particularly well in Fig. 265, Plate V, where the auricular wave, *a*, gradually approaches the carotid and ventricular wave till it is not discernible as a distinct wave. It was during periods such as this that Dr. Crump and I made out the

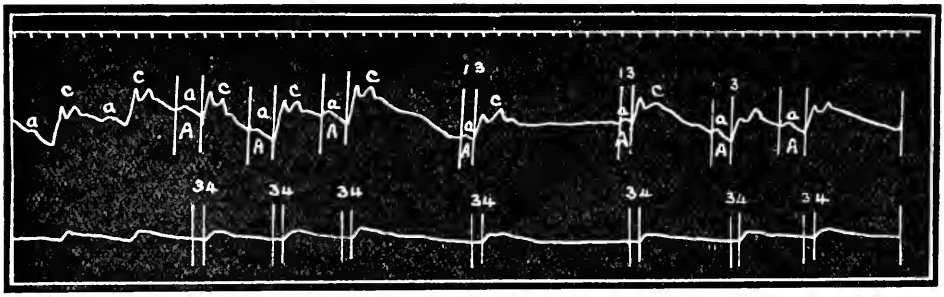


FIG. 274. The upper tracing was taken high up in the neck and shows a slight auricular wave (*a*) preceding the carotid pulse (*c*). After a long pause the *a–c* interval (space *A*) is much diminished. (Case 28.)

change in the relation of the presystolic murmur to the first sound and its apparent cessation—as shown by the shading underneath the jugular tracing.

It may be taken as certain that in this patient the marked slowing and long pauses were inhibitory in origin. But the inhibition appeared not to involve the a.-v. fibres, for their conductivity was not reduced, this proving that the drug acts on the a.-v. fibres through its action on the inhibitory function and not through its direct effects on the heart muscle. (Compare Case 27.)

Another phase that occasionally occurred during the irregular period was the appearance of extra-systoles. Figs. 266 and 267, Plate V (Fig. 267 is a continuation of Fig. 266), are characteristic examples, and show that the extra-systoles are probably of ventricular origin, though the pauses following the extra-systoles are of varying duration, due to the influence of the digitalis on the sinus.

This case, as I have said, is an exception to the general rule that when there is a delayed *a–c* interval digitalis increases it and produces blocking

of the systoles between auricle and ventricle. I have carefully considered whether there may not have been blocking between the sinus and auricle, as has been shown to occur by Wenckebach [327] and Hewlett [202]. I can find no reason for suspecting such a thing here, particularly as the slow periods have no such regularity as to lead one to infer that the sinus was beating at regular intervals. In this case the whole heart seemed affected by the digitalis, but on exceptional occasions the ventricles escaped and produced extra-systoles.

Records of blood-pressure showed generally a fall (100 mm. Hg.) when there was much dropsy, and a rise with slowing of the heart to 135 or 140 mm. Hg. Sometimes, however, the pressure was 130 with dropsy, and no increase occurred with its disappearance and the coincident improvement of the patient's condition after taking digitalis.

APPENDIX VII

The Electro-cardiogram *

By Thomas Lewis, M.D., M.R.C.P.

1. *The relation of contraction to electric changes in muscular tissue.*

For many years it has been known that muscular activity is accompanied by electric changes. When a strip of somatic musculature is stimulated at one end, and contraction is induced at the point of stimulation, this point becomes electro-negative to the resting or inactive end. If, as a result of the excitation, a wave of contraction is propagated in the muscle, then, as each successive segment enters upon the contractile state, each segment becomes relatively electro-negative. The change in potential, or the variation in the current produced by it, may be registered by connecting the two ends of the strip to suitable recording apparatus. In the instance of a wave of contraction which passes from end to end of a single strip, the changes registered are diphasic ; for at first the excited end is negative to the distal end, and eventually, as the distal end becomes negative, the excited end is relatively positive.

Now precisely analogous changes in electric state occur in visceral musculature, such as cardiac muscle, contracting under the influence of physiological impulses, and these may be similarly recorded. Activity is always associated with the development of negativity, and if leads are made from base and apex of the ventricle and the electric state of one relative to the other is recorded, it will be obvious *that a very definite idea of the direction which the contraction-wave takes in the ventricle may be formed. And, further, an important clue to the point at which contraction starts will be at our disposal.* These conclusions are of great importance, and should form the basis of all electro-cardiographic studies.

2. *Electric changes as a result of the heart beat.*

In the frog, electric changes as a result of the heart beat were described by Kölliker and Müller [451] in 1855 ; their observations were followed by those of many other workers. The mammalian heart was the subject of special investigation at the hands of Waller [457, 458, 459] and others. It was found that the type of curve yielded by leads from base and apex, is approximately duplicated by leads from other parts of the body. Stated in a simple form, it

* The expenses of the work, upon which this appendix is based, have been defrayed by the British Medical Association.

was shown that the body can be mapped out by a plane crossing the base of the heart, into two parts, one abutting upon the base, the other including the apex of the ventricle ; *and that leads from these two divisions of the body are equivalent to leads from base and apex of the ventricle.* Thus the possibility of registering the electric changes produced by the heart beat in the intact animal was demonstrated, and in man they were first studied by Waller. In the human subject, the dividing-line already referred to passes, according to the last-named writer, from the left shoulder to the right loin. The right arm may therefore be utilized as a basal lead, while the left arm, or either leg,* serves as an apical lead. Leading off from the human body, through

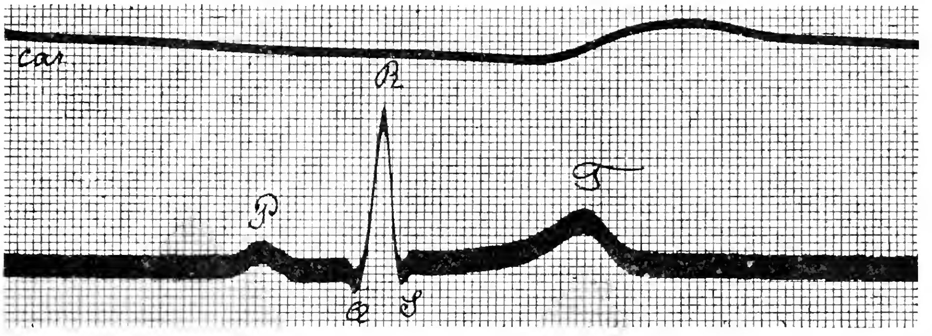


Fig. 275, for which we are indebted to Professor Einthoven, shows a portion of a carotid curve and a single beat of an electric curve. The abscissae are divided at intervals of 0·01 sec.; the ordinates are divided at intervals of 10^{-4} volt. The lead, as in all figures shown, was from right hand and left foot. In this, as in all figures also, corresponding points of time are directly vertical to each other. P represents the auricular and R, S, and T the ventricular contraction.

the unbroken skin, records of the electric change as a result of systole of the ventricle may therefore be obtained, and they correspond very closely to records yielded experimentally by direct leads from base and apex of the mammalian heart. *In addition, distinct changes as a result of auricular contraction are registered.*

The method employed clinically.

The galvanometer which is employed clinically is that invented by Einthoven [444, 445], to whose papers the reader is referred for a detailed account of its construction and working.

It consists of a heavy magnet, permanent or in circuit, the poles of which are close together. Between the poles a fine platinum or silvered quartz thread is suspended. When the instrument (string galvanometer) † is

* The changes registered necessarily vary to some extent with the ‘ lead ’.

† Sold by the Cambridge Scientific Instrument Co., and by Dr. Th. Edelmann of Münich.

working, the thread lies in a powerful magnetic field, and deviates whenever a current is led through it. The shadow of the string and its movements are magnified and projected, by means of a system of lenses and an arc light, on to a photographic apparatus. The delicacy of the instrument depends upon the strength of the magnetic field, and the fineness of the conducting thread. The body-current which it is desired to register is led from the limbs (right arm and left leg, or right arm and left arm) by immersing them in electrolytic solutions which are in connexion with the ends of the suspended thread.

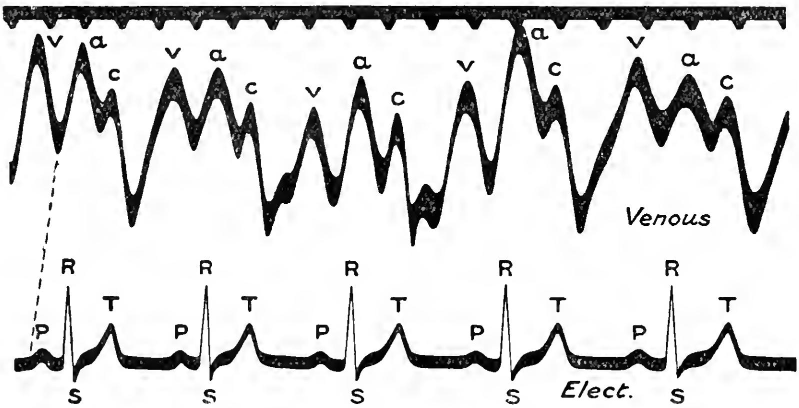


FIG. 276. Simultaneous records of time, in $\frac{1}{5}$ sec., venous and electro-cardiographic curves. The delay in the venous curve is due to the air transmission employed, to the delay in transmission from auricle to neck, and to the fact that the electric change slightly precedes the contraction. From a patient in which the P–R and *a–c* intervals show slight prolongation, but in which the curve is otherwise normal.

3. *The normal electro-cardiogram and its significance.*

The normal electro-cardiographic curve consists of three negative and usually one positive wave (directed upward and downward respectively in the accompanying figures). They are designated by the letters P, R, and T (negative peaks) and S (positive depression). P is a result of auricular contraction. R, S, and T are due to systole of the ventricle.

The ventricular portion of the curve (R, S, and T) is thus triphasic.* It

* Not infrequently the curve shows four phases, Q, R, S, and T (Fig. 275). The depression Q, which is distinct in Fig. 275, is usually less marked than the depression S, but may be more prominent than the latter. In brief, the opening events of the ventricular electro-cardiogram are subject to variations, and it is possible that these variations depend on slight differences in the direction of the contraction-wave when it starts in the ventricular musculature. It will simplify the description if further reference to this depression Q is omitted, for it is not certain that it is ventricular in origin. Nevertheless, its frequent presence should not be forgotten.

shows two displacements, R and T, in a direction indicating negativity of the base of the heart, and one displacement, S, indicating negativity of the apex (or positivity of the base). The curve is of such a nature as to suggest that the contraction of the heart originates at its base, travels to the apex, and returns to the base (Gotch [447]) (cf. § 1).

4. *Variations in the individual electro-cardiographic curve.*

Both auricular and ventricular portions of the galvanometer curve are subject to considerable variation in form, a variation seen more especially in pathological conditions. Thus in mitral stenosis an increase in the size of P accompanies hypertrophy of the auricle, and in the later stages of the affection, P shows a division into two parts (P_1 and P_2 of Fig. 277), and may be prolonged as a whole. (The normal P–R interval varies from ·12–·16 seconds ; the P–R interval in the accompanying figure, Fig. 277, is ·2 second ; in the normal curve, Fig. 275, it is ·15 second).

FIG. 277. From a case of mitral stenosis and aortic regurgitation. Showing the prolongation of P–R interval, a splitting of P, and an increase in S.

Similarly, variations are met with in the ventricular portion of the curve. Thus hypertrophy of the left ventricle, especially hypertrophy of its apical portions, such as is met with in aortic regurgitation, is commonly accompanied by an increase in the magnitude of the variation S. This fact, which is in accordance with the interpretation of the normal curve, is well illustrated in Fig. 277, and in the patient from which this curve was obtained left ventricular hypertrophy was a notable feature.

5. *Analysis of the sequence of contraction in the cardiac chambers.*

In the last paragraph examples were given illustrative of information to be obtained from individual auricular or ventricular curves, where the heart beat shows the usual sequence of chamber contraction. The method is of still greater service in the analysis of disordered sequence, and in this direction forms a valuable adjunct to venous pulse work.

For the time being two examples will suffice. Fig. 278 is taken from a case of complete heart-block. The photograph, as a whole, is composed

of frequent auricular and infrequent ventricular curves, accurately superimposed upon each other. The ventricular curve is identified by the peak R, its most constant feature. The variation T is anomalous and inverted (a by no means uncommon occurrence in pathological conditions). The peak P occurs at regular intervals in the figure, and whenever it falls with a ventricular curve it is superimposed upon it.

The second example is shown in Fig. 279, and is taken from a patient with the disorderly action of the heart, termed ' Nodal rhythm ' in this book. It shows no sign of the normal auricular contraction during the diastole of the heart. Further, it shows marked tremulousness of the line joining the

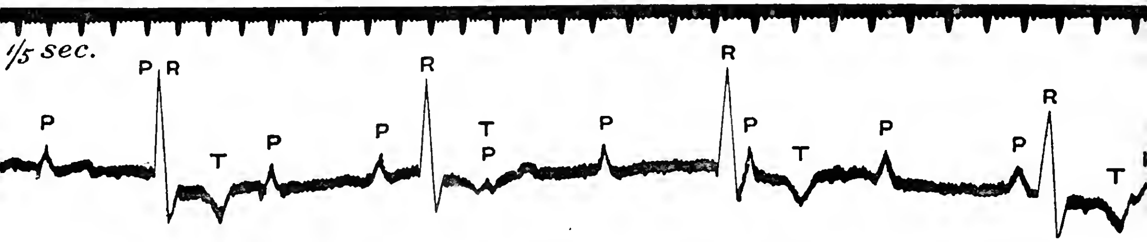


FIG. 278. From a case of complete heart-block, showing the dissociation of auricle and ventricle.

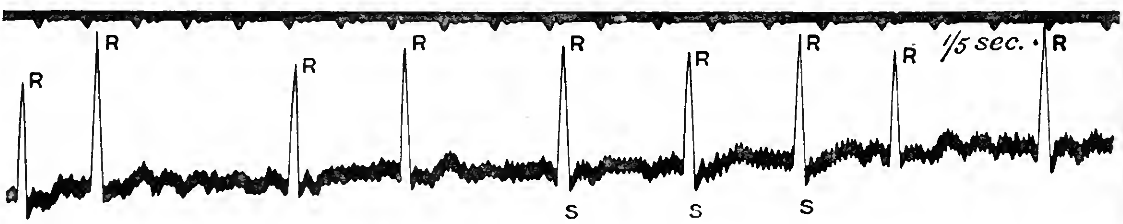


FIG. 279. From a case of ' nodal rhythm ', showing the absence of the normal wave P, and the presence of irregular waves having fixed relationship to other events.

peaks R. This tremulousness has been found in association with myocardial degeneration * (Einthoven, Kraus and Nicolai [452, 45]).

6. *The point in the musculature at which a contraction arises.*

In the preceding paragraphs the importance of electro-cardiographic curves have been shown from two separate aspects. First, they give information of the nature of a contraction in auricle or ventricle respectively. Secondly, they serve in the analysis of disordered rhythm and in the elucidation of disturbed sequence of systole. Information of the first and second

* The explanation of the tremulousness will be fully dealt with in an early article to *Heart*.

order is also obtained by other methods; by direct inspection, palpation or percussion, &c., and by means of graphic records from pulsating areas. The electro-cardiogram may consequently be said to yield, as a rule, evidence which is but confirmatory of that obtained by other means, when regarded from these special points of view. But on this account its importance should not be underrated, for it frequently happens that the remaining physical signs are obscure or difficult to obtain.

Finally, the galvanometer yields evidence of a distinctive nature. It allows recognition of contractions arising in the separate chambers of the heart, which start in areas of the musculature other than the normal. It is in this direction that galvanometric observations promise the richest harvest of fact. We have seen that the normal electro-cardiogram of the

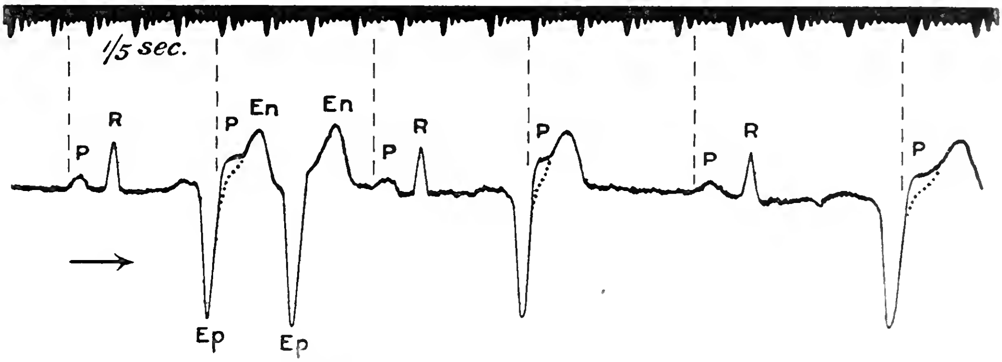


Fig. 280. The curve shows three beats in which the normal P–R sequence is observed, and four extra-systoles of the ventricle. The P waves which are regular are superimposed upon the extra-systolic curves in three places (dotted lines).

ventricle commences with a tall and sharply-pointed peak, R, and it has been stated that in the presence of this peak we have presumptive evidence of the origin of ventricular contraction in the morphological base of the ventricle. In examples of complete heart-block (Fig. 278), the ventricular curve commences in the same way, and we may conclude with a fair degree of certainty that the beats of the ventricle in this condition are propagated from a point in its musculature, corresponding to that from which they start in the normal beat. A similar observation applies to the curves of ‘nodal rhythm’ (Fig. 279). In this instance, as in that of complete heart-block also, it may be asserted that all the beats start in the ventricle from a single focus.

In Fig. 280 an example of ventricular extra-systoles is portrayed. The normal beats (of which three are present) are represented by P, R variations. The extra-systolic curves (of which four are present) are highly atypical. Each is manifested by a primary positive and a secondary

negative displacement. They are all of the same nature, and therefore arise from a single focus in the ventricular musculature. The actual point of origin is undecided, but there is evidence to show that they proceed from the left ventricle and rather from its apical than from its basal portion.* The ultimate elucidation of the birth-place of anomalous beats of a nature similar to those shown is a matter of experiment and time. It will be obvious from the remarks in § 1 that when a systole of the ventricle starts at a point other than the normal, such atypical curves must result, and that many forms of atypical curve are to be anticipated. Such is found to be the case, but at present a full description of these curves would not be profitable. Again, just as beats, arising in areas of the ventricular musculature other than the normal starting-points, produce atypical electrical curves, so also do those beats which arise out of place in the auricle. For example, when a beat of the auricle is started by a beat of the ventricle (retrograde contraction) the peak P disappears and is replaced by a curve of a highly atypical character.

The importance of this aspect of the electro-cardiogram will now be clear, for in all probability it will eventually be possible to accurately determine the focus in which the impulses of all such abnormal contractions are developed.

It occasionally happens that extra-systolic contractions of the ventricle fall at such points in the auricular cycles that it is impossible to determine by venous pulse methods whether such ventricular beats are the result of transmitted auricular impulses or not. In such cases the nature of the electro-cardiographic curve of these beats may be very helpful. In Fig. 280 the second auricular curve falls upon the first extra-systolic curve (it is indicated by a dotted outline). It is succeeded by a contraction of the ventricle, which in the absence of the galvanometric curve, might have been readily mistaken for a response to auricle. With the evidence of the electric curve before us, its origin in an auricular impulse can be definitely denied, and it is recognized as an extra-systole of precisely the same nature as those which precede and succeed it.

* Curves identical with those shown in the figure may be obtained by electrical stimulation of the heart apex.

FIG. 4. Respiratory movements from a case of Cheyne-Stokes respiration (see page 29).

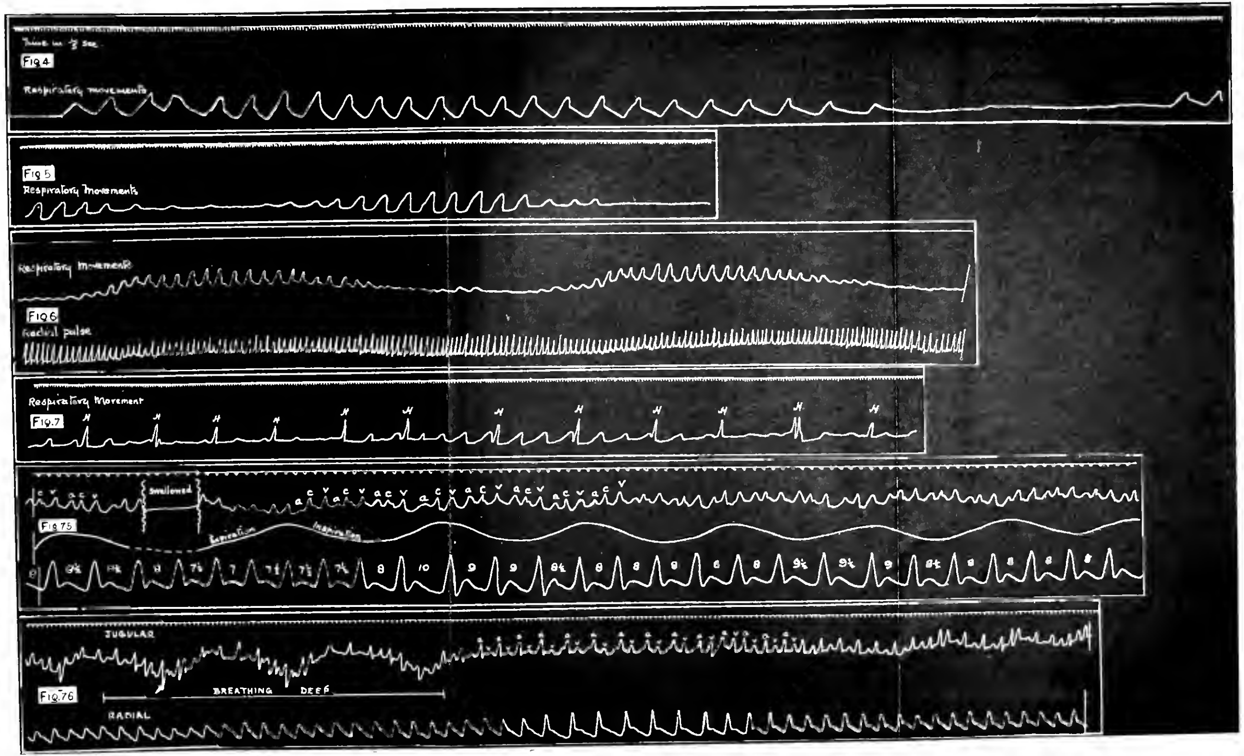
FIG. 5. Respiratory movements from a case of Cheyne-Stokes respiration (see page 29).

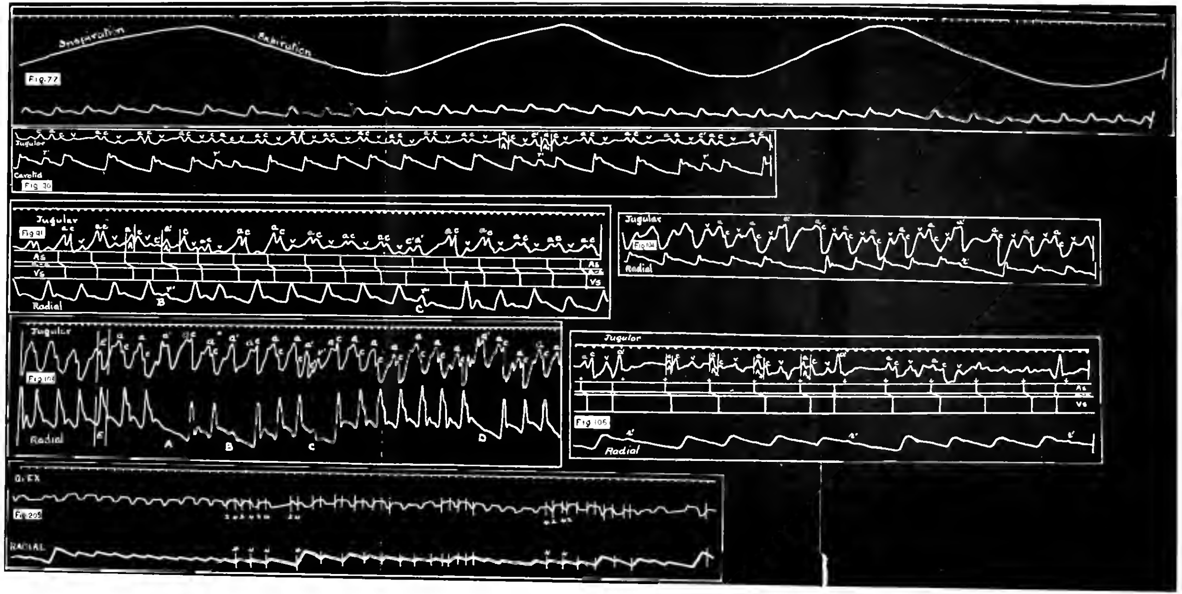
FIG. 6. Simultaneous tracings of the respiratory movements and the radial pulse from a patient with Cheyne-Stokes respiration. The base line in the radial tracing rises during the respiratory phase and the irregularity of the pulse (due to depression of contractility) becomes more marked. The blood pressure also rose at the same time. (Case 23, Appendix V; see page 29.)

FIG. 7. Tracing of the respiratory movements and of the movements due to Hiccough (*H*) during Cheyne-Stokes respiration (see page 30).

FIG. 75. Sinus irregularity produced by reflex stimulation of the vagus by the act of swallowing. The numbers refer to the duration of each cardiac cycle in tenths of seconds, and they show that the heart quickened its rate for a few beats after swallowing, then gradually slowed, then again increased slightly in rate, followed by another short period of slowing (9½ & 9). The respiratory curve shows that the irregularity was independent of respiration. Compare with Figs. 258 and 259, Plate IV (see page 144).

FIG. 76. Sinus irregularity (produced after three deep inspirations) shown by a temporary slowing of the pulse (see page 144).




Inspiration
Expiration
Fig. 77
Jugular
Carotid
Fig. 30
Jugular
Fig. 91
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Radial
Jugular
Fig. 104
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As
Vs
Fig. 105
Radial
Fig. 205
RADIAL

FIG.

FIG.

FIG.

FIG.

FIG.

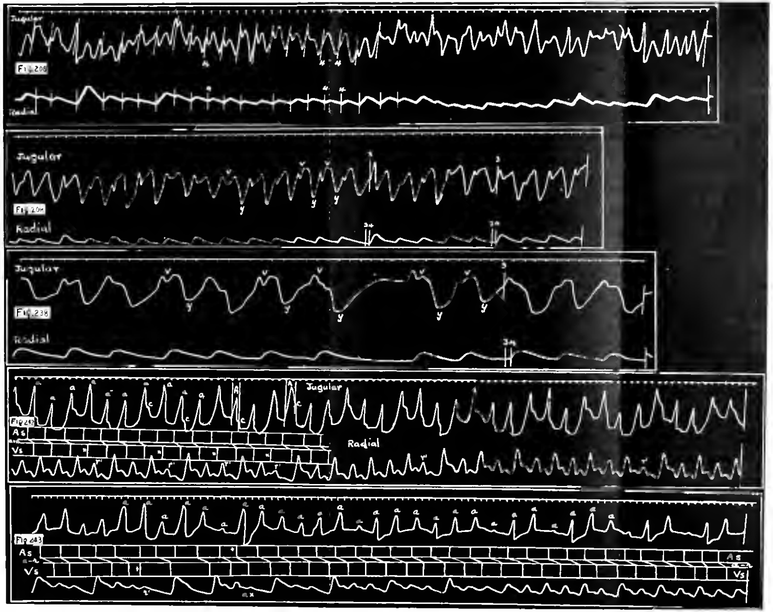


FIG. 206. Shows the characteristic change in the jugular pulse (ventricular form of jugular pulse) with the sudden inception of the nodal rhythm. (Case 10, July, 1906; see page 320.)

FIG. 208. After the heart's rate has become slower the irregularity and jugular pulse (ventricular form) characteristic of the nodal rhythm persist. (Case 10, November, 1906; see page 320.)

FIG. 238. Continued irregularity which has persisted from 1904 to 1908. The jugular pulse is of the ventricular form. (Case 17, 1908; see page [illegible].)

FIG. 242. Shows the pulsus alternans in the radial tracing, with the frequent occurrence of ventricular extra-systoles [illegible]. In the jugular tracing there is only one large wave, a, due to the auricle. There is [illegible] an impairment of conductivity of the a–c bundle, as shown by the variation of the a–c interval (space I). (Case 24; see page 354.)

FIG. 243. The time [illegible] in the interpolated diagram (a–c) represents the time between the [illegible] wave in the jugular and the radial pulse. There are ventricular extra-systoles [illegible] and auricular ones [illegible], but the auricular action is not regular, as shown by the variation in the As intervals, and the a–c intervals also vary, indicating, as in Fig. 242, an impairment of the conductivity of the a–c bundle. These two tracings therefore show evidence of impairment of the a–c bundle, and of depressed contractility of the left ventricle. (Case 24; see page 354.)

Fig. 244. The respiratory curve shows the heart's irregularity to be independent of the respiration. The trigeminal appearance of the radial pulse seems to be due to the occurrence of ventricular extra-systoles (x) and auricular (a') after the smaller wave of the pulsus alternans. (Case 24; see page 354.)

[illegible] 258. Shows the effect of the vagus due to swallowing. After the act of swallowing the pulse became very slow because of the dropping out of the ventricular systoles (see diagram in Fig. 259). After this the heart's rate increased slightly, then became [illegible] again. (Case 27; see page 362.)

[illegible] the same as Fig. 258, except that during the second period of slowing after [illegible], there is a long pause due to the dropping out of a ventricular systole (see [illegible]).

[illegible] the inception of the nodal rhythm after digitalis. Compare the jugular [illegible] the murmur with Fig. 272, page 367. (Case 28; see page 366.)

[illegible] 26[illegible] [illegible] of the whole heart for 19 fifths of a second and 15 fifths. Note [illegible] character of the tracing after the pause. Taken at the same visit as Fig. 2[illegible], page 367. Digitalis effect. (Case 28; see page 366.)

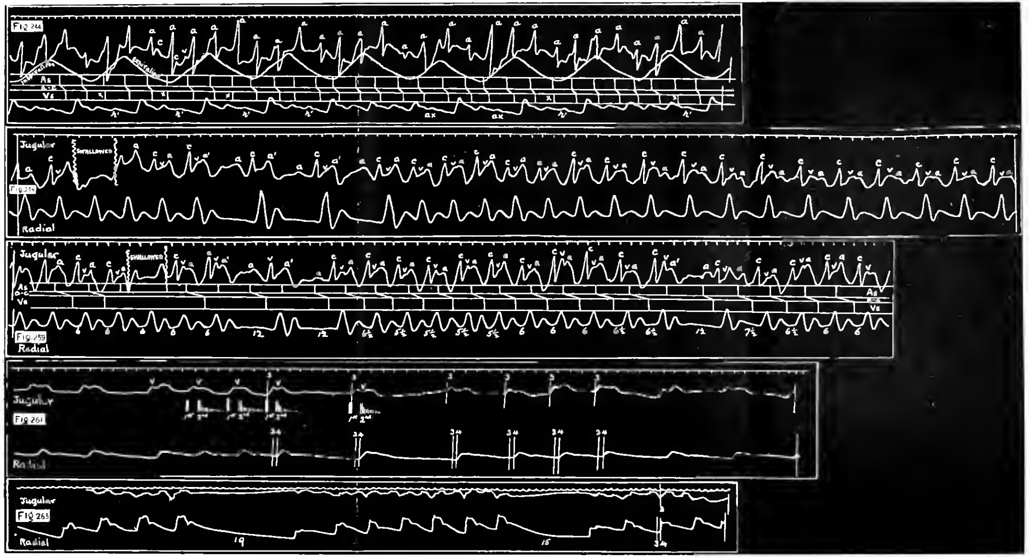
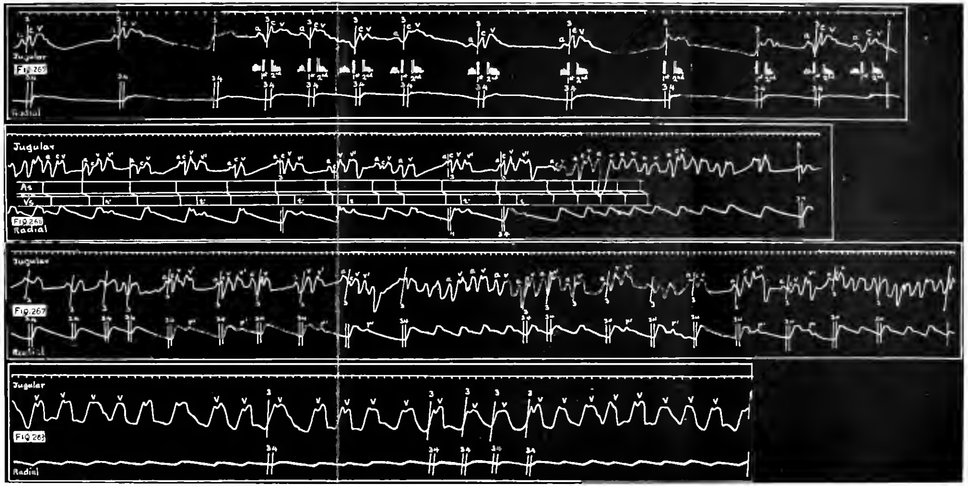


Fig. 265. Slowing of the whole heart due to digitalis. During the pauses the conductivity of the a-v bundle became so well restored that the auricular wave *a* gradually approached the carotid wave *c* till they became blended. The murmur due to the auricular systoles also approaches and seemed to become lost in the first sound as shown by the shading. (Case 28, see page 366.)

Fig. 266. Extra-systoles, *e*, probably of ventricular origin due to digitalis. The waves *e* are due to the extra-systole. There are occasional long pauses when no extra-systole occurred followed by a shortening of the *a-c* interval. (Case 28, see page 366.)

Fig. 267 is a continuation of Fig. 266 and shows the same kinds of irregularity. (Case 28, see page 366.)

Fig. 268. Shows the irregularity and ventricular form of venous pulse characteristic of the nodal rhythm which finally became permanent in this patient. (Case 28, see page 366.)



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